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Electromyographic Data and Post-Exercise Pain in Female Muscle Pain and Control Subjects after Experimental Chewing

by

Dr. Glenna M. Grykuliak ©

A thesis submitted to the Faculty of Graduate Studies and Research in partial
fulfillment of the requirements for the degree of
Masters of Science

in

Orthodontics

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled **Electromyographic Data and Post-Exercise Pain in Female Muscle Pain and Control Subjects after Experimental Chewing** by Glenna M. Grykuliak in partial fulfillment of the requirements for the degree of Masters of Science in Orthodontics.

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Abstract

This study examined (1) the postural EMG muscle activity directly following a chewing exercise in 27 female muscle pain subjects and 20 female non-pain subjects and (2) the relationship between EMG data and post-masticatory pain scores. The EMG activities of bilateral anterior temporalis and masseter muscles were measured before and after a 4 minute chewing exercise. Subjects rated their bilateral pre- and post-chewing pain intensity on a 100 mm visual analog scale. Although there was statistically significantly higher immediately post-chewing muscle activity in the left masseter muscle of pain patients compared to the non-pain subjects, the difference between the groups was small and lasted for a short duration. Thus, pain and nonpain female subjects had a similar recovery of baseline EMG activity following a 4 minute chewing exercise. A linear regression analysis of pre- and post-chewing EMG data and ipsilateral pre- and post-chewing pain scores showed that little of the variability in EMG data could be explained by variability in the pain scores. Thus, the results of this study do not provide support for the "muscle hyperactivity" theory.

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QUOTE PAGE

We have not wings, we cannot soar
    But we have feet to scale and climb
By slow degrees, by more and more,
    The cloudy summits of our time…

Henry Wadsworth Longfellow

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DEDICATION

To my dear husband, Chris,
for his faith in me.

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Table of Contents

Chapter 1 – Introduction and Literature Review

1.1...Introduction...2
1.2...Statement of the Problem...6
1.3...Purpose...9
1.4... Research Questions...10
1.5...Null Hypotheses...11
1.6...Defining Temporomandibular Disorders and Myofascial Pain...12
    .1...Defining Temporomandibular Disorders...12
    .2...Defining Myofascial Pain and Myofascial Pain-Dysfunction Syndrome...13
1.7...Defining Electromyography...15
1.8...Muscle Pain: A Major Symptom of TMD...15
    .1...Prevalence/Incidence of Muscle Pain...16
    .2...Theories on the Mechanism of Pain...16
        .a...Pain Due to Contraction Fatigue...17
            (1)...The Definition of Fatigue...18
            (2)...Fatigue and Endurance Time...18
            (3)...Fatigue and EMG Amplitude...19
            (4)...Fatigue and Power Spectrum Analysis...21
            (5)...Conclusion...22
1.9...Theories on the Etiology of Myogenous TMD...23
    .1...The Hyperactivity Theories...23
        .a...Postural Hyperactivity...23
            (1)...Studies Inducing Muscular Pain...24
            (2)...EMG Assessment Studies of Postural Activity...25
            (3)...Biofeedback Treatment...31
        .b...Muscle Hyperactivity During Chewing...34
            (1)...EMG Asymmetry During Mastication...35
        .c...Muscle Hyperactivity During Parafunction...37
        .d...Causes of Hyperactivity...39
            (1)...Local Causes of Hyperactivity...40
            (2)...*Centrally Mediated Causes of Hyperactivity...40*
                .i...Psychological Theories...41
                .ii...Vicious Cycle Theory...42
    .2...The Centrally Mediated Theory of TMD...43
1.10...Conclusion...43
Reference List for Chapter 1...45

Table of Contents (Continued)

Chapter 2 – Research Paper

- 2.1...Introduction...56
- 2.2...Materials and Methods...59
    - Subjects...59
    - Procedure...60
- 2.3...Statistics...63
    - EMG...63
    - Pain Ratings and EMG...63
- 2.4...Results...64
    - EMG...64
    - Pain Ratings and EMG...65
- 2.5...Discussion...66
- 2.6...Conclusions...72
    - Figures (2-1 to 2-4) of Mean Left Masseter EMG Activity vs Time (Pain and Non-pain)...73
    - Figures (2-5 to 2-8) of Mean Right Masseter EMG Activity vs Time (Pain and Non-pain)...74
    - Figures (2-9 to 2-12) of Mean Left Temporalis EMG Activity vs Time (Pain and Non-pain)...75
    - Figures (2-13 to 2-16) of Mean Right Temporalis EMG Activity vs Time (Pain and Non-pain)...76
    - Figures (2-17 to 2-18) of Differences Between Immediate Post-Chewing EMG and Baseline EMG in the Masseter Muscles vs Time (Pain and Non-pain)...77
    - Figures (2-19 to 2-20) of Differences Between Immediate Post-Chewing EMG and Baseline EMG in the Temporalis Muscles vs Time (Pain and Non-pain)...78
    - Table 2-1. VAS Data: Changes in Pain Intensity After Exercise...79
    - Table 2-2. Results of Linear Regression on EMG and Pain Scores in Muscle Pain Subjects...80
- Reference List for Chapter 2...81

Chapter 3 – Discussions and Recommendations

- 3.1...General Discussion...87
- 3.2...Recommendations for Future Studies...99
- Reference List for Chapter 3...101

Appendices...105

List of Tables

Table 2-1 VAS Data: Changes in Pain Intensity After Exercise…79

Table 2-2 Results of Linear Regression on EMG and Pain Scores in Muscle Pain Subjects…80

Table 3 Appendix Item #11. P-Values of Left Masseter Mean EMG (Pain and Non-pain)…117

Table 4 Appendix Item #12. P-Values of Right Masseter Mean EMG (Pain and Non-pain)…118

Table 5 Appendix Item #13. P-Values of Left Temporalis Mean EMG (Pain and Non-pain)…119

Table 6 Appendix Item #14. P-Values of Right Temporalis Mean EMG (Pain and Non-pain)…120

List of Figures

Figure 2-1 Left Masseter Prior to Chewing…73
Figure 2-2 Left Masseter Immediately After…73
Figure 2-3 Left Masseter 1 Min After…73
Figure 2-4 Left Masseter 3 Min After…73
Figure 2-5 Right Masseter Prior to Chewing…74
Figure 2-6 Right Masseter Immediately After…74
Figure 2-7 Right Masseter 1 Min After…74
Figure 2-8 Right Masseter 3 Min After…74
Figure 2-9 Left Temporalis Prior to Chewing…75
Figure 2-10 Left Temporalis Immediately After…75
Figure 2-11 Left Temporalis 1 Min After…75
Figure 2-12 Left Temporalis 3 Min After…75
Figure 2-13 Right Temporalis Prior to Chewing…76
Figure 2-14 Right Temporalis Immediately After…76
Figure 2-15 Right Temporalis 1 Min After…76
Figure 2-16 Right Temporalis 3 Min After…76
Figure 2-17 Left Masseter: Immed After – Baseline…77
Figure 2-18 Right Masseter: Immed After – Baseline…77
Figure 2-19 Left Temporalis: Immed After – Baseline…78
Figure 2-20 Right Temporalis: Immed After – Baseline…78
Figure 21 LM (Ses 1) Prior to Chewing…113
Figure 22 LM (Ses 1) Immediately After…113
Figure 23 LM (Ses 1) 1 Min After…113
Figure 24 LM (Ses 1) 3 Min After…113
Figure 25 RM (Ses 1) Prior to Chewing…114
Figure 26 RM (Ses 1) Immediately After…114
Figure 27 RM (Ses 1) 1 Min After…114
Figure 28 RM (Ses 1) 3 Min After…114
Figure 29 LT (Ses 1) Prior to Chewing…115
Figure 30 LT (Ses 1) Immediately After…115
Figure 31 LT (Ses 1) 1 Min After…115
Figure 32 LT (Ses 1) 3 Min After…115
Figure 33 RT (Ses 1) Prior to Chewing…116
Figure 34 RT (Ses 1) Immediately After…116
Figure 35 RT (Ses 1) 1 Min After…116
Figure 36 RT (Ses 1) 3 Min After…116
Figure 37 LM (Ses 1) Immed After – Baseline…121
Figure 38 RM (Ses 1) Immed After – Baseline…121

List of Figures (Continued)

Figure 39 LT (Ses 1) Immed After – Baseline...121
Figure 40 RT (Ses 1) Immed After – Baseline...121

List of Abbreviations and Symbols

CMD - craniomandibular disorders

EMG - electromyography, electromyographic

kΩ - killiohm(s)

LM - left masseter

LT - left temporalis

mm - millimeter(s)

MPD - myofascial pain-dysfunction or myofascial pain-dysfunction syndrome

MPF - mean power frequency

MVC - maximum voluntary contraction

MVOF - maximum voluntary occlusal force

RM - right masseter

rms - root mean square

RT - right temporalis

TM - temporomandibular

TMD - temporomandibular disorders or temporomandibular joint-dysfunction syndrome

TMJ - temporomandibular joint

VAS - visual analog scale

µV- microvolt(s)

List of Appendices

Item #1: Information Sheet…105
Item #2: Consent Form…107
Item #3: Questionnaire (Page 1)…108
Item #4: Questionnaire (Page 2)…110
Item #5: Pre-exercise Pain Score…111
Item #6: Post-exercise Pain Score…112
Item #7: Figures of Session 1 Mean Left Masseter EMG Activity vs Time (Pain and Non-pain)…113
Item #8: Figures of Session 1 Mean Right Masseter EMG Activity vs Time (Pain and Non-pain)…114
Item #9: Figures of Session 1 Mean Left Temporalis EMG Activity vs Time (Pain and Non-pain)…115
Item #10: Figures of Session 1 Mean Right Temporalis EMG Activity vs Time (Pain and Non-pain)…116
Item #11: Table 3. P-Values of Left Masseter Mean EMG (Pain and Non-pain)…117
Item #12: Table 4. P-Values of Right Masseter Mean EMG (Pain and Non-pain)…118
Item #13: Table 5. P-Values of Left Temporalis Mean EMG (Pain and Non-pain)…119
Item #14: Table 6. P-Values of Right Temporalis Mean EMG (Pain and Non-pain)…120
Item #15: Session 1 Differences Between Immediate Post-chewing EMG and Baseline EMG in the Masseter and Temporalis Muscles vs Time (Pain and Non-pain)…121

Chapter 1

Introduction and Literature Review

1.1...Introduction

The role of a dentist is to maintain the general health of the masticatory complex. Patients who experience pain in their mouth or associated facial structures during speech, chewing, and rest are often in great distress. Pain in the masticatory complex can be due to a variety of causes. The origins of masticatory pain may include the dentition, the supporting tissues of the dentition, the temporomandibular joint, the muscles of mastication, and other facial structures such as the sinuses. By diagnosing the cause of the pain, the dentist is able to treat the patient or to refer the patient to the appropriate health care provider for treatment.

Many patients present to their dentists and medical doctors with complaints of symptoms involving their temporomandibular joint (TMJ). They may be told that they suffer from TMJ dysfunction syndrome or a temporomandibular disorder. The term temporomandibular disorder (TMD) is more commonly used today than TMJ dysfunction syndrome (Rugh and Solberg, 1985). The term describes a variety of signs and symptoms. One significant symptom is masticatory muscle pain, which may or may not be associated with other signs and symptoms.

Masticatory muscle pain may be treated by a variety of methods that include the placement of an intraoral appliance, pharmacotherapy with medications such as analgesics and muscle relaxants, the application of cold and heat, therapeutic exercises, electrostimulation, and external biofeedback (dos Santos, Jr., 1995). Additional treatment modalities include behavioral treatments such as massed practice, alarm systems, and habit reversal (Gevirtz *et al*, 1995). Not all of these treatment modalities are appropriate to every patient with masticatory muscle pain. For example, due to its vasoconstrictive effect, the application of cold is a suitable treatment for a patient suffering from a recent direct trauma to a muscle (dos Santos, Jr., 1995). However, cold application is not as useful in treating a patient suffering from muscle pain due to a bruxism habit. In this case, an intraoral appliance might be more effective. While some dentists have sufficient knowledge to use many of these modalities to treat a patient with masticatory muscle pain, most dentists are limited to using appliances and medications. Dentists may refer their patients to a physiotherapist for instruction in the use of heat and cold, therapeutic exercises, and electrostimulation or to a psychologist for external biofeedback and behavioral treatments.

There are several modes of external biofeedback. The most commonly used type is electromyographic (EMG) biofeedback (dos Santos, Jr., 1995). EMG biofeedback-assisted relaxation is a form of cultivated low arousal (Gevirtz *et al*, 1995). The purpose of EMG biofeedback-assisted relaxation is to detect muscle activity and to display the electrical activity of a given muscle in either an audio or visual signal, which the patient is made aware of. This awareness helps the patient learn how to control the firing of muscle motor units (dos Santos, Jr., 1995). Another form of cultivated low arousal is stress management (Gevirtz *et al*, 1995).

Gevirtz *et al* (1995) described the techniques used by therapists in cultivated low arousal. Treatment usually ranges from six to twelve sessions. The most common sites for EMG biofeedback-assisted relaxation are the masseter, anterior temporalis, and frontal areas. Patients learn how to position their jaws to minimize EMG activity in these muscles. Besides biofeedback, patients are also trained in relaxation procedures and stress management.

Some patients may benefit from a multimodal psychophysiological stress profile. This assessment often includes the measurement of EMG, finger temperature, skin conductance, heart rate, and respiration rate during stressors and recovery after stressors. Such stressors could include counting backwards by sevens from a large three-digit number or imagining a personal stressor for three minutes. By showing the patients patterns of reactivity and recovery, the patients may learn to decrease their reactions to the stressor and to decrease their recovery time. This psychophysiological profile has its origins in research done by Yemm.

Yemm (1969c) compared masseter muscle activity during experimental stress between a group of normal subjects and TMD patients. Unlike the control subjects, the TMD subjects' EMG activity did not decrease when the stressors were repeated three times. Yemm labeled this finding in the TMD patients as a failure of adaptation and hypothesized that it might contribute to the etiology of their TM disorder. Yemm (1969a) also suggested that if the muscular activity during experimental stress was higher, it was possible that the muscle activity was increased even when the TMD subjects were not performing the tasks.

In a review of the scientific evidence for biofeedback, Mealiea and McLynn (1987) found many studies that show that biofeedback is an effective treatment for TMD. However, they did criticize many of the studies for their lack of control for placebo effects and for the presence of confounding variables. Due to these problems with research protocol, the authors concluded that they found no definitive experimental support for the use of biofeedback in TMD patients. The authors did note that the application of biofeedback to TMD patients seemed to be no worse than other treatment techniques. For example, Turk *et al* (1993) found that while splint therapy was more effective in reducing pain than biofeedback/stress-management techniques on a short-term basis, a six-month follow-up indicated that the appliance group experienced considerable relapse while the biofeedback/stress-management patients were continuing to improve.

Researchers still do not have a clear understanding of how biofeedback works. Gevirtz *et al* (1995) outlined two main theories on the mechanism of biofeedback. First, it was hypothesized that biofeedback works by decreasing EMG activity, which then reduces the patient's pain. This is not a generally accepted theory because investigators have found that a decrease in EMG activity did not correspond directly to a decrease in pain symptoms (Peck and Kraft, 1977; Dahlstrom *et al*, 1984; Burdette and Gale, 1988). Second, it was suggested that biofeedback helps the patient develop general relaxation skills and improved coping strategies as well as an improved body awareness. In support of this theory, Glaros (1996) found that TMD patients have deficits in proprioceptive awareness during nonstress periods when compared with nonpain subjects.

It is imperative that any dentist who treats TMD patients be well-educated in all possible treatment modalities for this group of disorders. By gaining a thorough understanding of masticatory muscle EMG and EMG biofeedback, the dental practitioner becomes better prepared for helping to alleviate the masticatory muscular pain associated with TMD.

1.2...Statement of the Problem

Yemm's research (1969a,b,c) has contributed to the psychophysiological theory (Laskin, 1969) of hyperactivity. This theory states that psychological stress causes a centrally mediated hyperactivity response in the muscles of mastication (Laskin, 1969; Yemm 1969a,b,c; Rugh and Solberg, 1976). The psychophysiological hyperactivity theory has received support in studies where experimentally-induced stress has produced a significantly greater increase in masticatory muscle activity of TMD patients compared with controls (Yemm, 1969c; Mercuri *et al*, 1979; Katz *et al*, 1989). In a study similar to that of Yemm (1969c), Katz *et al* (1989) found a small but significant difference between the right temporalis muscle activity of TMD patients and control subjects during stressful stimuli. However, the lack of statistically significant differences in the EMG activity of bilateral masseter and the left temporalis muscles caused the authors to question the clinical significance of their findings. In recent years, there has been considerable criticism of the hyperactivity theories. Gervais (1984) and Katz *et al* (1989) compared EMG activity during and after stressors and found that there was no difference between TMD patients and nonpain subjects during the recovery period. Katz *et al* and Gervais are among the few researchers who report the post-stressor EMG response. Most of the research in this area focussed only on the EMG activity during a stressor. Other authors found inadequate experimental evidence that hyperactivity was linked to TMD (Moss and Garrett, 1984; Lund and Widmer, 1989; Lund *et al*, 1991).

The question of the effect of physical stressors, such as mastication, is also an interesting one because of the association between mastication and pain. Some TMD subjects reported that chewing or mastication caused or aggravated facial pain (Moss *et al*, 1984; Glass *et al*, 1993). The self-reported prevalence of pain on chewing in TMD patients varied from approximately 37% (Zarb and Thompson, 1970; Fricton *et al*, 1985; Dworkin *et al*, 1990) to 68% (Bush *et al*, 1989). When TMD patients were selected specifically for having pain in the masticatory muscles, the self-reported prevalence of pain on chewing varied from 67.2% (Dao *et al*, 1994) to 81% (Bush *et al*, 1989). An increase in postexercise muscle pain was reported in patients with fibromyalgia/myofascial pain (Jacobsen *et al*, 1993). Dao *et al* (1994) found that a three minute chewing test increased pain intensity in about half (48.3%) of a group of 60 TMD patients suffering from muscle pain. An unusual finding by these investigators was that about one-third (31.7%) of the pain subjects experienced a decrease in their pain symptoms after chewing.

While there have been many studies done on the EMG activity during mastication, the literature does not yield a significant amount of research on the postexercise EMG activity of the muscles of mastication. Tzakis *et al* (1994) measured muscle activity before and after a thirty minute chewing exercise. The pre- and post-chewing recordings were comprised of the postural EMG activity and the activity during maximal clenching in the intercuspal position. They found no significant difference in EMG activity between the experimental group and control group. One criticism of the study is that the researchers did not indicate the amount of time that elapsed between the cessation of mastication and the EMG recordings. Second, the experimental group consisted of only healthy subjects in whom pain was achieved through vigorous mastication. Clark *et al* (1989) concluded that a healthy subject experiencing pain is not a good model for a TMD subject in a study involving isometric contraction. It is possible that this may also be the case in studies involving mastication.

The postexercise response of TMD subjects could be of use to therapists using biofeedback. Gevirtz *et al* (1995) described the use of a psychophysiological profile to assess a patient's EMG activity both during the psychological stressor and during their recovery after the stressor. If a patient had a slow rate of recovery to the psychological stressor, the patient was taught to reduce the amount of time it took to return to their resting baseline EMG activity. Approximately half of a sample of TMD patients reported pain following mastication (Dao *et al*, 1994). Based on this finding, it would be useful to observe the recovery of baseline EMG activity in TMD patients compared with nonpain subjects following a physical stressor, such as mastication. A significant difference might be useful to biofeedback therapists, who may then guide their patients to be aware of and to modify their post-exercise muscle activity, thereby reducing pain.

1.3...Purpose

This study will test the "muscle hyperactivity" theory by observing if female TMD patients have statistically significantly different post-masticatory muscle activity than female non-pain subjects and if there is a positive correlation between the EMG activity and pain scores recorded after a chewing exercise. First, the EMG muscle activity directly following a chewing exercise will be compared between female TMD patients with muscle pain and non-pain females. Only female subjects were chosen because females report more frequently for treatment of TMD than males by a ratio of approximately 3:1 (Rugh and Solberg, 1985). Second, this study will assess whether there is any relationship between post-masticatory EMG levels and post-masticatory self-reported pain scores in pain subjects.

1.4...Research Questions

1. Is there a statistically significant difference between the postural EMG activity during the recovery period following a chewing exercise in female TMD patients who have masticatory muscle pain and female non-pain subjects?

2. Is there a statistically significant correlation between the subjective magnitude of pain of the female TMD patients and the postural EMG activity following a chewing exercise?

1.5...Null Hypotheses

1. There is no statistically significant difference between the postural EMG activity during the recovery period following a chewing exercise in female TMD patients who have masticatory muscle pain and female non-pain subjects.

2. There is no statistically significant correlation between the subjective magnitude of pain of the female TMD patients and the postural EMG activity during the recovery period of a chewing exercise.

1.6...Defining Temporomandibular Disorders and Myofascial Pain

1.6.1...Defining Temporomandibular Disorders

A collective term for pathology in the masticatory system is craniomandibular disorders (CMD). A synonymous term is temporomandibular disorders or TMD (Rugh and Solberg, 1985; Okeson, 1996), which is now the most commonly used term. A third and less frequently used name found in the literature is stomatognathic dysfunction syndrome (Abekura et. al, 1995). Temporomandibular disorders are defined according to signs and symptoms experienced by the patient (Dahlstrom, 1989). They are described as a group of disorders that are characterized by pain and tenderness in the masticatory muscles and/or temporomandibular joint (TMJ), or the preauricular area of the face. Other signs and symptoms include sounds during condylar movement, and limitation of mandibular movement (Dahlstrom, 1989). The mandible may move asymmetrically during function (Okeson, 1996).

There is controversy in the literature about the definition and classification of TMD. Bakke and Moller (1992) subdivide CMD into temporomandibular joint-dysfunction syndrome (also shortened to TMD) and myofascial pain-dysfunction syndrome (MPD). According to these authors, the dental occlusion is thought to be the etiology of pathology in the joints seen in TMD. In MPD, stress-related and emotional reactions are thought to be the cause of pathology confined to the masticatory muscles. This categorization of CMD into TMD and MPD according to etiologies is not accepted by all researchers in this area of study. However, the benefit of this categorization is that it emphasizes the concept that either the joints or muscles may be predominant in temporomandibular disorders. For example, if a patient is diagnosed with

TMD, it may be that she has no symptoms of joint pathology while she may be experiencing considerable discomfort in her jaw muscles.

1.6.2...Defining Myofascial Pain and Myofascial Pain-Dysfunction Syndrome

The terms myofascial pain and myofascial pain-dysfunction syndrome (MPD) are utilized frequently in research. A review of the literature yielded two major definitions of myofascial pain. The American Academy of Orofacial Pain gives the first definition to myofascial pain in an addendum to the Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias, and Facial Pain of the International Headache Society. According to their description, the two key diagnostic features of myofascial pain are a dull, aching pain and localized trigger points in muscle, tendon, or fascia (Okeson 1996). A characteristic pattern of referred pain and/or autonomic symptoms may result from palpation of these trigger points (Okeson, 1996). The key to this definition of myofascial pain is the presence of trigger points. The second definition of myofascial pain is much more general. Dao *et al* (1994) use the term myofascial pain to simply mean pain in the muscles of mastication and their associated tendon, or fascia.

Many authors who use the term MPD, described this syndrome in a variety of ways. The definition of MPD given by Chaconas and Fragiskos (1990) was very similar to the definition of TMD, which encompassed joint and/or muscle symptoms (Dahlstrom, 1989). Other authors described MPD as a muscle facial pain disorder that is linked to stress and muscular tension (Bakke and Moller, 1992, Dolan and Keefe, 1989, Erlandson and Poppen, 1989). In a review of papers, Erlandson and Poppen (1989) found that MPD was described as a stress-related, functional muscle disorder that can lead to organic changes in the TMJ. They suggested that hyperactivity in the muscles of mastication is an important etiological factor in MPD. It is thought that this elevation of muscular activity levels, as measured by EMG, occurred before and after mandibular movement. The result of this hyperactivity is a specific type of masticatory muscle pain on jaw movement which has been described as discomfort, tiredness in the head and face, and muscular tenderness. It is a controversial definition because not all researchers agree that muscle hyperactivity, as measured by EMG, is common feature of TMD patients with muscle pain (Lund and Widmer, 1989, Lund et. al., 1991).

Okeson (1985) provided a definition of MPD as masticatory muscle spasms or cyclic myospastic activity. Okeson (1985) proposed that MPD begins as muscle splinting, which is a protective mechanism used by the muscles to restrict movements of damaged structures. When the splinting continues uncontrolled, the pain caused by the muscle splinting feeds back to the muscle. The pain experienced with this spasm can increase the activity of the gamma efferents, which in turn increases muscle activity.

The purpose of the presentation of these different definitions of myofascial pain and MPD is to illustrate the confusion that exists in the literature. It is obvious that standardization of nomenclature is of utmost importance. For the purposes of this literature review, the term TMD will encompass all muscular and/or joint disorders. To differentiate between muscle and joint symptoms, the terms myogenous TMD and arthrogenous TMD will be used (De Leeuw *et al*, 1994; Reid *et al*, 1994).

Instead of referring to the muscle pain that TMD patients experience as myofascial pain (Dao et. al., 1994), it will be described simply as muscle pain or facial pain. The term myofascial pain will be used solely to refer to those individuals who are diagnosed as having trigger points (Okeson, 1996).

1.7...Defining Electromyography

The study of myogenous TMD has been facilitated by the use of EMG. Electromyography recording directly measures muscle activity and the integrated EMG signals reflect the chewing force almost linearly (Kumai, 1993). Thus, EMG is a measure of muscle recruitment. Electromyography can detect muscle hyperactivity and hypoactivity, spasm, fatigue, and muscle imbalance (Lund and Widmer, 1989). Electromyographic activity varies with the age, gender, and facial morphology of the subject (Lund and Widmer, 1989). For example, EMG activity generated in a standard isometric contraction has been shown to decrease in amplitude with increasing age (Carlsson, Alston, and Feldman, 1964). In addition, surface EMG of females is of higher amplitude than that of males lifting same weight (Visser and De Rijke, 1974). The proposed explanation for the gender difference is that women must recruit a higher number of motor units at a higher frequency in order to produce the same contractile force as a man (Visser and De Rijke, 1974).

1.8...Muscle Pain: A Major Symptom of TMD

The most frequent presenting symptom of TMD is pain (Moss and Garrett, 1984). This pain usually occurs in the muscles of mastication, the preauricular area, and/or the TMJ (Okeson, 1996). The pain is most often reported to be unilateral and the quality of pain is usually a dull ache (Moss and Garrett, 1984).

The lateral pterygoid muscle most frequently found to be tender and painful (Moss and Garrett, 1984).

1.8.1...Prevalence/Incidence of Muscle Pain

There is controversy about whether signs and symptoms of TMD are more common in women than in men (Okeson, 1996). The use of a nonspecific overall symptom level (eg, Helkimo index) in non-patient surveys indicated that there was little difference between men and women (Agerberg and Carlsson, 1972). However, when muscle tenderness was evaluated separately, there was a greater reported incidence in women (Agerberg and Bergenholz, 1989).

Studies of patients seeking treatment for TMD reported that 30% to 41% had a masticatory muscle disorder (Schiffman *et al*, 1989; Pullinger and Seligman, 1991). However, of the people reporting TMD symptoms, it is estimated that only 3.6% to 7% of these patients require treatment (Okeson, 1996).

1.8.2...Theories on the Mechanisms of Pain

Moss and Garrett (1984) outlined the historical evolution of the theories on the cause of muscle pain. First, Costen (1934) hypothesized that muscle pain and joint pain were caused by overclosure of the mandible resulting in the placement of pressure by the TMJ on the auriculotemporal and chorda tympani nerves. This theory was challenged by Zimmerman (1951), who stated that Costen's theory was anatomically improbable. Schwartz (1955) proposed that masticatory muscle spasm was responsible for pain. Yemm (1976) disagreed with Schwartz because he did not observe a difference between right and left masseter activity when only one of the muscles was tender. Instead, he proposed that the muscle pain was a result of damage to a part of the muscle.

No strong clinical evidence has been found to refute this theory (Moss and Garrett, 1984). Christensen (1975) suggested that increased fluid pressure in the muscle following sustained muscle contraction might be the cause of pain. To support this theory, Berry and Yemm (1974) used infrared to determine that the skin over a tender area of the masseter was hotter than the same spot on the opposite side. They attributed this heat to an increased blood flow. Other researchers (Jow and Clark, 1989) have proposed that pain was caused by a decrease in post-contraction blood flow. Further explanations for muscular pain include morphologic and metabolic changes related to muscle contraction such as a decrease in the muscle pH, an increase in the inorganic-phosphate-to-creatine-phosphate ratio, and an increase of intramuscular pressure (Dao *et al*, 1994). Muscle tenderness may be related to prolonged central hyperexcitability and altered CNS processing following peripheral tissue damage or nerve injury (Reid *et al*, 1994). This theory was supported a study by Reid *et al* (1994), who found that muscle pain had poor localizability and was diffuse in nature. A final theory is that pain is caused by fatigue of the muscles of mastication. This theory will be discussed in greater detail because of the use of EMG data to investigate this theory.

1.8.2.a...Pain Due to Contraction Fatigue

Researchers have suggested that the fatigue resulting from muscle hyperactivity during rest and excess function, such as clenching or bruxism, could cause some of the pain associated with TMD (Bakke and Moller, 1992; Jacobs and van Steenberghe, 1993). The research that supports and challenges this theory will be reviewed in the following sections. The major argument against this fatigue theory of pain is the finding that the masticatory muscles may be relatively fatigue-resistant.

1.8.2.a(1)... The Definition of Fatigue

Fatigue has been described as an inability of the neuromuscular system to produce a continuous level of force or work (Bigland-Ritchie, 1981a). In the studies that have been done to date, three general methods have been used to measure fatigue. These include the study of endurance time, the measure of EMG amplitude, and the evaluation of the EMG power spectrum (Mao *et al*, 1993).

1.8.2.a(2)...Fatigue and Endurance Time

Maximal voluntary isometric contractions (MVC) or maximum voluntary occlusal force (MVOF) of the elevator jaw muscles, with the mandible in the position of maximal intercuspation of the natural teeth, can induce fatigue as well as severe pains in the jaw muscles of children and adults (Christensen, 1981). Christensen and Mohamed (1984) found that muscle fatigue occurred after 30 seconds of clenching in healthy asymptomatic subjects. However, 30 seconds of grinding, which consisted of combined concentric and eccentric contractions, induced no fatigue. The onset of fatigue was defined as a subjective sensation by the subject. This sensation was associated with an increase in myoelectrical signals, measured in microvolt-seconds (μV.s), followed by a drop in mean voltage (μV) of the masticatory muscles. At the onset of fatigue, it was hypothesized that most of the motor units had been recruited. This was followed by a progressive decline in isometric tension as indicated by the drop in mean voltage (Christensen and Mohamed, 1984).

Other investigators reported similar endurance times to that of Christensen and Mohamed (1984) for subjects who were clenching with MVOF (Palla and Ash, 1981; Dahlstrom *et al*, 1988). A variety of investigators found that endurance times were greater, ranging from 53 seconds to 15 minutes when only 40% to 50% MVOF was used (Naeije, 1984; Hellsing and Lindstrom, 1983). Jow and Clark (1989) studied endurance times in ten healthy males with rest periods between clenches and they found that endurance times for clenching depended on the duration of rest periods in between clenches. They also found that endurance time was always greater for the first clench relative to all subsequent clenches.

One of the challenges in measuring the endurance time in masticatory muscles is the dependence on the subjective reporting of discomfort and pain. Although isometric contractions were shown to cause the onset of jaw muscle pain, it was hypothesized that the perception of the moment of onset of pain depended on a large but unknown extent on psychological factors (Christensen, 1981). Thus, the EMG changes at the onset of patient-reported fatigue were considered an important adjunct to the study of fatigue and pain (Christensen, 1981). A second criticism of these studies is that there are different definitions of the failure point (Mao *et al*, 1993). Some investigators define the failure point as the time when the subject has no more power to clench while others define it as the onset of discomfort or pain. In order to eliminate these problems, the use of EMG in determining the onset of fatigue has been studied.

1.8.2.a(3)...Fatigue and EMG Amplitude

Contraction fatigue is caused either by an inability to convert the extracellular electrical transmission into an intracellular chemical reaction or by a failure in the chain of intracellular reactions (Mao *et al*, 1997 . It is manifested either by a drop in the force while the amplitude of the EMG activity remains the same or increases (Haraldson *et al*, 1985) or by an increase in EMG during an endurance test (Christensen and Mohamed, 1984). However, Hellsing and Lindstrom (1983) found that when 40% incisal MVOF was sustained, the EMG activity decreased in the masseter while it increased, as expected, in the temporalis muscle.

Other researchers challenged the idea that jaw elevator muscles are susceptible to contraction failure. They made this conclusion when they found the MVOF and the EMG contraction levels did not change following an endurance exercise (Clark and Carter, 1985; Clark and Adler, 1987). Clark and Carter (1985) asked their subjects to maintain various submaximal occlusal forces (25% MVOF, 50% MVOF, and 75% MVOF). During these submaximal tasks, the subjects were asked to perform brief 100% MVOF. The authors found that the brief MVOF values and corresponding brief EMG contraction levels did not change during or after these fatigue-inducing submaximal tasks. This was true even shortly after the subjects reached their endurance limit, which was defined as the onset of pain. Since Clark and Carter (1985) hypothesized that rapid recovery was not consistent with contraction failure, they concluded that contraction failure had not occurred. Instead, they attributed the endurance limit to pain intolerance.

Many of the studies cited above included only healthy male subjects (Christensen and Mohamed, 1984; Clark and Carter, 1985; Clark and Adler, 1987; Jow and Clark, 1989). Clark *et al* (1989, 1991) hypothesized that healthy male subjects performing sustained isometric clenching did not serve as good models for chronic or subacute muscle pain subjects because of insignificant pre- and post-MVC differences between jaw pain, active opening, and maximum lateral excursions. It is possible that TMD patients would not react to sustained isometric contraction in the same way as healthy subjects. Some important information might have been gained if these studies had included subjects with a history of TMD.

1.8.2.a(4)...Fatigue and Power Spectrum Analysis

Power spectrum analysis has been used to study fatigue. To understand power spectrum analysis, one must first have an understanding of muscle fiber types. Each muscle is composed of different fiber types. Mammalian muscle fibers are categorized into type I, type IIA, and type IIB fibers based on histochemical studies (Brooke and Kaiser, 1970). Type I fibers are red or aerobic fibers that produce sustained low-level forces in tasks such as maintaining posture, and are fatigue-resistant providing oxygen is available (Basmajian and De Luca, 1985). The type II fibers are white, anaerobic, and are more prone to fatigue (Basmajian and De Luca, 1985). Human jaw muscles differ from other mammalian muscles in that the type II fibers are smaller than the type I fibers (Nordstrom and Miles, 1990). The human masseter is composed mainly of the type I fibers (Eriksson and Thornell, 1983).

These different types of fiber in a muscle have different activation rates. Axons that supply slow muscle fibers (i.e. type I fibers) have a slower conduction velocity and discharge rate than axons supplying the fast type II fibers. An EMG recording of a muscle contains these frequencies which relate to the activation rates. A mathematic calculation of the EMG record derives a spectrum of the different frequencies (Basmajian and De Luca, 1985). From this spectrum, the average frequency can be calculated. This is known as mean power frequency (MPF). The expected finding is that the MPF shifts to a lower value as the muscle fatigues (Lund and Widmer, 1989) and some studies have shown this (Palla and Ash, 1981; Clark *et al*, 1988; Jow and Clark, 1989; Jacobs and van Steenberghe,1993). This is because the low frequency, fatigue-resistant components increase and the high frequency, more fatigable components decrease (Junge and Clark, 1993). Other explanations for this shift are that it is caused by a decrease in firing rate (Bigland-Ritchie *et al*, 1979) and a slowing of muscle fiber conduction velocity (Lindstrom *et al*, 1985).

The advantage of using power spectral analysis is that it provides an objective fatigue parameter (Jacobs and van Steenberghe, 1993). Problems using this method include variations in the frequency spectrum of each masticatory muscle, presence of shifts in patients without fatigue, and no reported shifts in patients with fatigue (Lund and Widmer, 1989). Also, it has been questioned whether a MPF shift is a true representative of contraction fatigue (Clark *et al*, 1988, Junge and Clark, 1993). Clark *et al* (1988) and Junge and Clark (1993) showed that the sum of the EMG amplitude of the masseter and anterior temporalis muscles divided by the occlusal force magnitude (EMG/force ratio) remained constant during sustained contractions at various levels of force. However, the MPF of these muscles did drop. Junge and Clark (1993) utilized another quantitative measure of EMG activity, the number of turns or reversals of direction. They found a constant turns/force ratio during and after a sustained contraction. From these findings, the investigators concluded that the jaw elevator muscles are fatigue-resistant.

1.8.2.a(5)...Conclusion

Due to the controversy in this area of study, it cannot be concluded that muscle hyperactivity due to excess postural tension and/or excess function causes fatigue. The main argument against this theory is that the muscles of mastication are relatively fatigue-resistant. However, the variability in results and conclusions from the research on fatigue indicate a need for further study. It has been suggested that future research could include the study of all members of a synergistic muscle group in order to detect overcompensations by some muscles for decreased activity in one member of the group (Hellsing and Lindstrom, 1983). For example, the medial pterygoid activity has been rarely studied due to the need for needle electrodes to measure the muscle's EMG (Mao *et al*, 1993).

1.9...Theories on the Etiology of Myogenous TMD

There are many theories on the cause of muscle pain in TMD patients. For this discussion, these theories will be divided into two major categories: the hyperactivity theories and centrally mediated theories. The formulation and study of these theories is of great importance. If the cause of the muscle pain in TMD patients is known, it follows that treatment for this disorder will be made easier.

1.9.1...The Hyperactivity Theories

Many researchers theorize that muscle hyperactivity plays an integral role in the etiology of muscle pain in TMD patients (Dahlstrom, 1989; Bakke and Moller, 1992). In addition to measuring postural muscle activity, muscle hyperactivity has been studied during normal muscle activities, such as chewing, and parafunctional muscular activities, such as clenching and bruxism.

1.9.1.a...Postural Hyperactivity

In their literature review, Moss and Garrett (1984) found three ways that postural hyperactivity in facial pain has been studied: 1) studies inducing muscular pain in the muscles of mastication in non-TMD subjects; 2) EMG assessment studies comparing EMG activity in the muscles of TMD and non-TMD subjects; 3) studies employing treatments, such as biofeedback designed to reduce masticatory muscle activity.

1.9.1.a(1)...Studies Inducing Muscular Pain

These studies have been reviewed previously, under the heading of 1.8.2.a...Pain Due to Fatigue (Contraction Fatigue). In the fatigue studies, healthy subjects were usually asked to clench their teeth in order to induce pain. In some studies such as Christensen and Mohamed (1984), changes in the level of absolute EMG activity were recorded at the subject's endurance limit. In other studies, power spectral analysis was used to quantify any changes in EMG activity at the onset of fatigue. While some investigators showed a change in mean EMG activity (Christensen and Mohamed, 1984) and a downward shift in the mean power frequency (Jacobs and Van Steenberghe, 1993), other authors found no changes in these parameters (Clark and Carter, 1985).

Tzakis *et al* (1994) differed from the majority of investigators studying fatigue by inducing TMD symptoms by having subjects chew gum intensely for thirty minutes instead of clenching. They measured the effects of fatigue on the masticatory muscles of healthy subjects before and after 4 weeks of daily 1 hour chewing training. While marked clinical signs of dysfunction were recorded after the first fatigue test, EMG measures in the postural position and during maximal clenching did not differ significantly before and after the exercise. This study supports the hypothesis that the muscles of mastication are extremely fatigue-resistant (Clark and Carter, 1985).

The conclusion that the elevator muscles of the mandible are fatigue-resistant has been generally accepted. However, there are numerous criticisms of the fatigue studies, which have been discussed earlier. A compelling reason for doing further research in this area is that induced pain in healthy subjects may be a poor laboratory model for TMD patients experiencing non-induced pain (Clark *et al*, 1989). Thus, investigators have used alternative methods to study muscular involvement in TMD.

1.9.1.a(2)...EMG Assessment Studies of Postural Activity

There have been many studies comparing resting muscle activity in pain and nonpain subjects. There is significant disagreement among researchers as to whether experimental evidence of postural muscle hyperactivity in TMD subjects exists. Authors have reviewed the body of research in this area and presented their conclusions (Moss and Garrett, 1984; Dahlstrom, 1989; Lund and Widmer, 1989; Bakke and Moller, 1992). Dahlstrom (1989) and Bakke and Moller (1992) conclude that the studies consistently show an increased resting muscle activity in mandibular elevator muscles of TMD subjects when compared with control subjects. Lund and Widmer (1989), however, concluded that this hypothesis was unproven and that the variability in the subjects invalidated the use of postural EMG activity as a diagnostic parameter. These authors came to this conclusion after arguing that the majority of EMG studies did not control for subjects' ages, sex, facial morphologies, or histories of bruxism. Thus, Lund and Widmer (1989) found no convincing evidence to support the use of EMG in the diagnosis or treatment of dental patients, except in the treatment of parafunction.

Lund and Widmer (1989) discounted the postural hyperactivity theory because of their criticisms of many of the scientific methods used in the research. For example, they disagreed with the conclusions of Lous *et al* (1970). Lous *et al* showed an increased level of postural activity in the temporalis and masseter muscles of TMD patients (30 women, 9 men) relative to members of the control group (19 women, 26 men). When matched for gender, the differences in the masseter muscles disappeared while the differences in the temporalis muscles were still statistically significant between the pain and nonpain groups. Lund and Widmers' (1989) criticisms of this study include a poorly matched control group, inadequate blinding of the EMG analyst, and statistically significant, but small, differences in muscle activity. Bakke and Moller (1992) considered the age range differences between the study (14-70 years) and the control (20-30 years) groups to be relatively unimportant as the majority of study subjects were young (22 patients: 14-24 years).

Dohrmann and Laskin (1978) conducted a study similar to that of Lous *et al* (1970). However, Dohrmann and Laskin measured only the masseter muscle activity of their subjects. The investigators found a significantly higher level of postural activity in the pain subjects (21 women, 3 men; 20-71 years) relative to their controls (6 women, 1 man; 24-42 years). Again, Lund and Widmer (1989) criticized the poor matching of the study and control groups by Dohrmann and Laskin.

Majewski and Gale (1984), contrary to the authors of the previous studies, did not find a difference between the resting postural activity of pain and nonpain subjects. Bakke and Moller (1992) counter these findings by pointing out that the investigators asked their subjects to relax during the baseline recordings. It has been found that relaxation may reduce the enhanced postural activity in pain paitents (Moller *et al*, 1971).

Rugh and Montgomery (1987) recorded increased absolute postural activity in the masseter muscles of facial pain patients compared to a matched sample of controls. The mean resting baseline EMG levels of the patients (5.1 μV) versus the controls (2.2 μV) was maintained throughout the experiment, which involved the performance of stressful tasks. Katz *et al* (1989) also asked their subjects to perform stressful tasks. While there was a statistically significantly higher EMG activity in the right temporalis in the TMD subjects during stressors, the researchers did not find a significant difference between TMD and non-pain subjects of their resting EMG levels during all baseline recordings (prior to and in between psychomotor tasks).

Dolan and Keefe (1988) also compared postural muscle activity of the masseter muscles in muscle pain and non-pain subjects. Their patients were divided into groups having right-sided (13 patients) versus left-sided (18 patients) symptoms based on interview data. They showed that patients in the right-side-symptom group had twice the value of resting muscle activity on the left as they did on the right. However, there was no corresponding increase in the postural EMG activity of the right masseter in the left-sided-symptom group. In addition, the investigators further divided their subjects using their mean ratings of pain during the evaluation session. The pain was rated at the start and at the end of the evaluation session using a visual analog scale (VAS). They found that the left masseter activity was high across all conditions for patients in the high-pain, right-sided-symptom group (8 patients). However, the left masseter activity was not significantly increased in the low pain, right-sided-symptom group (10 patients). Thus, the authors concluded that the localization of symptoms to one side of the face and the reported pain level might be important factors in determining the postural muscle tension of a muscle pain patient. Majewski and Gale (1984), contrary to Dolan and Keefe found no significant difference of EMG levels between the pain and nonpain sides. Majewski and Gale compared the mean resting EMG activity levels of the anterior temporalis of patients with unilateral anterior temporal pain instead of the masseter muscle activity used by Dolan and Keefe.

Another criticism by Lund and Widmer (1989) is that the prevalence of bruxism may affect resting muscle activity. They cite Sherman's (1985) study that measured resting EMG activity of four groups: 1) subjects with TMD as well as a history and evidence of bruxing and clenching; 2) subjects with only TMD; 3) subjects with a history and evidence of bruxing and clenching; 4) subjects with no pain. Sherman found that subjects with a history of bruxing and clenching, with or without signs of TMD, had significantly higher resting EMG levels than all other subjects. In addition, after treatment using muscle tension awareness and relaxation, the majority of subjects with bruxing/clenching habits showed significant reduction in pain while the two other groups of subjects showed no significant improvement. Sherman concluded that bruxism and clenching were the causes of increased muscle activity.

Sherman was unclear in his description of how he assessed subjects for bruxism and clenching. He only indicated that they were divided into this group if there was "physical evidence" of a history of clenching and bruxism. The best current methods for assessment of parafunctional habits are portable electromyography, sleep laboratory, and direct observation (Okeson, 1996). None of these methods were used in Sherman's study. Thus, the significance of bruxism/clenching on resting EMG activity levels is not certain.

Additional criticism of studies of involving postural activity by Lund and Widmer (1989) involved the method of measurement. Some researchers compared "relative" resting activity rather than "absolute" resting EMG levels (Sheikholeslam *et al*, 1982). Michler *et al* (1988) reported both the absolute and "relative" resting activity. "Relative" resting activity is calculated as a percentage by dividing postural activity by mean maximal voluntary activity (Lund and Widmer, 1989). This "relative" measurement can also be used when expressing EMG activity during chewing and swallowing. The use of "relative" activity eliminates the variation that results from electrode positioning (Michler *et al*, 1988). In addition, this measure summarizes physical muscular strain better than absolute values (Bakke and Moller, 1992). Lund and Widmer (1989) oppose the use of relative postural activity because it has been shown that the EMG levels during MVC are generally higher in control subjects than in TMD subjects (Sheikholeslam *et al*, 1982; Bakke and Moller, 1992). Thus, differences in "relative" resting activity could be due to lower maximum output rather than a higher baseline (Lund and Widmer, 1989). It is important to report the absolute EMG values should the researcher decide to compare "relative" resting activity.

The most significant problem in drawing conclusions from this research is the significant variability between the studies. First, there was little standardization of the EMG apparati used in the studies. Second, there was variability in how the EMG activity was reported. For example, some investigators studied relative versus absolute EMG activity. Third, there was variability in the definition of the study and control groups. For example, some studies evaluated TMD patients (including arthrogenous TMD subjects) while others only included patients with muscle pain. Fourth, some studies had poorly matched of their study and control groups. Fifth, there was variability in the instruction given to the subjects during recording sessions. Finally, most studies did not control for bruxism due to the difficulty in assessing the presence of this parafunctional habit. All of these factors, and more which are not listed, may have contributed to the variability in results and the resulting diverse conclusions.

Despite the many criticisms discussed, there is some evidence to support the theory of postural hyperactivity mandibular elevator muscles in pain patients (Lous *et al*, 1970; Dohrmann and Laskin, 1978; Rugh and Montgomery, 1987). However, other research provides little support for the use of resting EMG data in accurately distinguishing facial pain patients from nonpain controls (Lund and Widmer, 1989; Glaros *et al*, 1997). Glaros *et al* (1997) tested the hypothesis that a cutoff score based on EMG values could be used to accurately separate TMD from non-TMD subjects. They scanned the EMG activity of the left and right frontalis, temporalis, and masseter muscles in fifty-four TMD and fifty-four non-TMD subjects who were matched for age and gender. Despite the fact that the TMD subjects had higher EMG activity in three of six sites examined, the application of a cutoff value resulted in a misclassification of approximately one third of the TMD and non-TMD subjects. Therefore, it does not seem feasible to assign a certain resting value as a "norm" for the elevator muscles.

This does not, however, preclude the use of postural EMG data. DeLuca (1988, c.f. Jankelson, 1990) stated that EMG data is valuable for evaluating the effectiveness of a given therapy. Jankelson (1990) concluded that the significant data collected in a clinical setting is the linear data from a single patient, which does provide diagnostic information for that particular patient.

1.9.1.a(3)...Biofeedback Treatment

Biofeedback treatment can be defined as treatment that employs electromyographic equipment to inform patients of their internal physiological events in the form of visual and/or auditory signals. The signals are used to teach patients to manipulate their internal physiological events by manipulating the visual or audible signals (Basmajian, 1983). Biofeedback treatment is used to treat a variety of muscle-related disorders, including myogenous TMD. The underlying premise of biofeedback is that lowering postural tonicity of muscles is desirable (Jankelson, 1990). The basic assumption underlying EMG biofeedback therapy is that muscle hyperactivity is the cause of pain and dysfunction (Moss and Garrett, 1984). From this assumption, one could predict that if EMG biofeedback therapy is successful, decreased EMG levels should accompany a decrease in symptoms.

Many of the biofeedback studies have shown improvement in TMD patients' symptoms with EMG biofeedback techniques (Gessel, 1975; Carlsson and Gale, 1977; Dohrmann and Laskin, 1978; Gale, 1979; Flor and Birbaumer, 1993; Turk *et al*, 1993). Dohrmann and Laskin (1978) concluded that the diurnal parafunction (daytime clenching/bruxism) in a group of MPD patients decreased by observing a post-treatment reduction in masseter EMG levels. However, these investigators did not do a long-term follow-up on their subjects. Gale (1979) measured the decrease in parafunction by establishing if subjects were symptom-free following treatment. He found that 80% of the experimental subjects were symptom-free immediately following treatment and 38% of the experimental subjects were symptom-free after 1 year. It was unclear if EMG activity was decreased in parallel with the reduction of symptoms. Other studies show a lack of correlation between a decrease in

EMG and a decrease in symptoms (Peck and Kraft, 1977; Dahlstrom *et al*, 1984; Burdette and Gale, 1988). If the theory that muscle hyperactivity is the cause of pain and dysfunction (Dahlstrom, 1989) is accurate, then it follows that when muscle activity is decreased, the pain and dysfunction should also decrease.

The addition of other therapies to EMG biofeedback has added to the confusion in biofeedback studies. For example, Gessel (1975) used biofeedback with general relaxation therapy and did not separate the effects. Turk *et al* (1993) combined biofeedback with stress management. This included didactic education, training in cognitive coping skills, and homework assignments to help patients practice relaxation without biofeedback. Again, the investigators did not separate the effects of biofeedback and stress management.

Some investigators have tried to control for the effects of augmenting biofeedback with other stress-reducing methods. Flor and Birbaumer (1993) compared the efficacy of EMG biofeedback, cognitive-behavioral therapy, and conservative medical interventions in 21 TMD patients. Cognitive behavioral therapy included instruction in pain and stress management, progressive muscle relaxation, and practice of pain-coping skills. At post-treatment, the biofeedback group showed the most significant improvements. At 6- and 24-month follow-ups, only the biofeedback group maintained significant reductions in pain severity, interference, affective distress, pain-related use of the health care system, stress-related reactivity of the affected muscles, and an increase in active coping self-statements. The authors concluded that patients suffering from musculoskeletal pain problems may benefit most from short-term biofeedback. Stenn *et al* (1979) found different results when they studied the masseter muscle activity of 11 TMD subjects. Six of these subjects received biofeedback and five were given relaxation training. All subjects had reduced muscle activity after their training and all reported a significant decrease in the number of symptoms. The authors concluded that both masseter EMG biofeedback and cognitive behavior modification were effective treatments for TMD.

The results of the biofeedback studies are controversial. Mealiea and McGlynn (1987) concluded that there was no definite support for the use of biofeedback-assisted masseter relaxation training alone or in combination with other treatments due to a lack of adequate research protocols in the biofeedback studies. The authors pointed out that it might be the research protocols and not the inefficacy of biofeedback that has led the inconclusive research. Lund and Widmer (1989) were critical of the biofeedback studies due to small sample sizes, lack of placebo groups, and lack of longitudinal monitoring.

Mealiea and McGlynn (1987) did recommend the combination of biofeedback with progressive relaxation training and/or behavioral counseling for patients with TMD. They supported their recommendation by pointing out that the results in biofeedback studies are comparable to data collected on other accepted TMD treatment techniques. For example, Turk *et al* (1993) found that while splint therapy was more effective in reducing pain than biofeedback/stress-management techniques on a short-term basis, a six-month follow-up indicated that the appliance group experienced considerable relapse while the biofeedback/stress-management patients were continuing to improve.

1.9.1.b...Muscle Hyperactivity During Chewing

Some investigators claim that muscle hyperactivity in natural chewing is an important factor in the development of pain-dysfunction symptoms. Stohler and Ash (1986) gave TMJ patients who reported sharp pain sensations in the joint region three pieces of Beefstick™ to chew. The patients used a thumbswitch to mark any event of discomfort or pain that was experienced during the chewing. The authors found that statistically significant prolonged contraction times and greater root mean square (rms) peak voltage amplitudes occurred for cycles where there was an indication of the experience of pain. The authors did not use a control group. In a subsequent study, Stohler *et al* (1988) found that there was significantly greater EMG activity in the jaw-closing muscles of TMD patients during the opening phase of painful masticatory cycles than in the pain-free cycles.

Dao *et al* (1994) showed that mastication exacerbates pain in subjects with “myofascial pain”. Pain was assessed before and after chewing in asymptomatic subjects and patients with muscle pain. Self-reports of pain were obtained with a checklist and on five-point category scales at the screening visit. Afterward, pain intensity at rest and after chewing wax for 3 minutes were reported on 100 mm visual analogue scales (VAS). They found that no control subjects had pain before or after chewing. However, approximately 50% of the MPD patients reported an increase of pain after chewing while the pain intensity decreased significantly after chewing in about 30% of the patient sample. The authors concluded that two subgroups of myofascial pain patients might exist with opposite reactions to exercise.

In summary, the studies indicate that mastication elicits pain in TMD subjects and that there are changes in EMG activity of the jaw-opening muscles associated with pain.

1.9.1.b(1)...EMG Asymmetry During Mastication

A finding which has recently been of interest to investigators is that the presence of TMD, a preference for unilateral mastication, and the asymmetry of masticatory muscle activity appear correlated (Kumai, 1993; Abekura *et al*, 1995). Unilateral mastication may impair the symmetrical activity of masseter muscles, leading to various disturbances of the stomatognathic system (Abekura *et al*, 1995).

From studies on healthy subjects, it has been established that there is a normal, predictable muscular pattern during mastication. Moore (1993) evaluated the EMG activity of bilateral mandibular muscle pairs in healthy subjects during mastication, voluntary oscillation of the jaw, and speech production. The subjects chewed hard candy on the right and left sides. The author classified the paired comparisons into homologous pairs (i.e. synchrony of activity in right masseter with left masseter), ipsilateral synergists (i.e. right masseter with right medial pterygoid), contralateral synergists (i.e. right masseter with left medial pterygoid), ipsilateral antagonists (i.e. right masseter with right digastric), and contralateral antagonists (i.e. right masseter with left digastric). He found that during mastication, homolgous pairs and ipsilateral synergists were coactivated to a degree significantly greater than either of the antagonist groups or the contralateral synergists. Using normal asymptomatic subjects, Balkhi *et al* (1993) investigated the reproducibility of EMG parameters descriptive of unilateral chewing, including activity, timing, and curve symmetry. The subjects chewed gum unilaterally for 10 second intervals. The EMG data was recorded three times (both right and left sides) in five separate sessions to study the reproducibility. The investigators found that EMG activity is more reliable than timing and EMG curve asymmetry. They also found that there was a unique order of activation of muscle activity during the initiation of mastication. In addition, the muscle activation order for the right side was considerably different from the left side, which suggested that pooling data for right and left side might be inappropriate.

Abekura *et al* (1995) demonstrated that the severity of symptoms of stomatognathic dysfunction increased as unilateral mastication increased. However, the investigators did not outline the method they used to determine the preferred chewing side. Similar to Abekura *et al*, Kumai (1993) concluded that the rate of unilateral chewing was greater in patients with unilateral TMD. In contrast to Abekura *et al*, he found a statistically significant difference between paired muscle activity during clenching in TMD patients when compared to normals. An interesting finding was that the preferred side in unilateral mastication was not necessarily ipsilateral to the side of dysfunction. This is in contrast to Ramfjord and Ash (1983) who reported that most patients with unilateral TMJ pain chew on the involved side, probably because this puts less pressure on the working condyle. Kumai (1993) determined his subjects' chewing patterns by evaluating EMG patterns during mastication of a peanut.

Christensen and Radue (1985) used a visual method to determine the lateral preference of normal subjects during chewing. They found that subjects who reported a preferred chewing side showed a significantly higher incidence of combined consistent and predominant bolus placement on the side preferred for chewing. Other methods of determining lateral preference for mastication include kinesiography (Neill, 1982; Howell *et al*, 1992), electromyography (Kumai *et al*, 1993), and cineradiography (Wictorin *et al*, 1971). The results of chewing studies may be affected by the variety of foods that are masticated. Chewing behavior is influenced by the nature of the test-food (Ahlgren, 1966). For example, bilateral chewing occurred more frequently with bread than with toffee (Wictorin *et al*, 1971).

Other factors may affect the asymmetry of muscle activity. First, maxillofacial morphology may affect the symmetry of muscular activity. For example, a person who has one masseter with a greater cross-sectional area will have greater muscle activity in an EMG recording. Second, occlusal factors such as interferences may influence muscular activity. Bakke and Moller (1980) found that significant asymmetry of muscle activity could be detected when premature contacts were evident. Third, physiological facial asymmetry exists. Some difference in the EMG activity between right and left muscles has been accepted as normal physiologic asymmetry (Abekura *et al*, 1995). Thus, although there appears to be a relationship between unilateral mastication, asymmetric muscle activity during mastication, and pain in TMD subjects, further research is required for this relationship to be well-supported with clinical evidence.

1.9.1.c...Muscle Hyperactivity During Parafunction

Bruxism is considered as a form of microtrauma because it causes sustained and repetitious adverse loading of the masticatory system (Okeson, 1996). This parafunctional habit is defined as nonfunctional jaw movements that include clenching, grinding, gritting, gnashing, and clicking of the teeth. Bruxism may also include contact of the teeth when one is chewing his cheeks, lips, pencils, or a pipe and it may lead to destructive changes in one or more components of the masticatory system (Hudzinski and Walters, 1987). The conditions which may result from bruxism are TMJ pain, tooth mobility, excessive tooth wear, destruction of restorations, and degenerative joint disease (Ware and Rugh, 1988). Bruxism may be done consciously or subconsciously and is thought to be an emotionally based activity (Hudzinski and Walters, 1987). Bruxism may be related to stressful life occurrences (Solberg *et al*, 1979; Clark *et al*, 1980). However, Pierce *et al* (1995) recently questioned this relationship when they failed to find a significant relationship between EMG activity (measures of bruxing frequency and duration) and personality or between EMG data and self-reported stress.

The reported prevalence of bruxism is variable. Signs and symptoms related to bruxism are found in up to 78% of the population (Solberg *et al*, 1979). A subsequent study reported only 31% of a group of over 1000 dental patients are past or current bruxers (Glaros, 1981). In a literature review, Dao *et al* (1994) found reports of 43% to 50% of "myofascial pain" patients self-report bruxism while 27% to 66% of TMD patients report a bruxism habit. The reason for this variability is unclear but it may be explained by the difficulty in diagnosing bruxism.

Investigators have used a variety of methods to determine if patients are bruxing. The diagnosis is often made on the presence of wear facets, soreness of jaw muscles upon awakening, sore teeth, and reports from the bed-partner (Lund and Widmer, 1989). Other investigators have also used the subjects' self-awareness of bruxism as a criterion. There are problems with these methods of assessing bruxism. For example, self-reports of bruxism are too subjective to use as an objective measurement. The problems with using attrition as a measure of bruxism were previously discussed. More research using portable

EMG and sleep laboratories are needed before the role of parafunction in TMD is clarified (Okeson, 1996).

Bruxism is a form of muscle hyperactivity. For example, Clark *et al* (1980) found that bruxers had greater nocturnal EMG activity than nonbruxers. However, the result of this hyperactivity is controversial. Clark *et al* (1981) found that a little- or no-TMD symptom group of subjects exhibited a significantly lower nocturnal EMG level than mild to moderate- and high-pain subjects. From this study, the authors concluded that there was a relationship between bruxism and TMD. This parafunctional habit has been implicated as an initiating or perpetuating factor in certain groups of TMD patients (Moss *et al*, 1984; Okeson, 1996). In addition, experimentally induced parafunction has been shown to cause pain similar to the pain reported to subjects with TMD (Christensen, 1981 and 1984). However, parafunctional habits do not always result in TMD symptoms (Okeson, 1986). Furthermore, it has been shown that clenching is not responsible for fatigue of the muscles of mastication (Clark and Carter, 1985; Clark and Adler, 1987).

1.9.1.d...Causes of Hyperactivity

Many researchers have proposed theories to explain the cause of hyperactivity in TMD patients. In this review, the hyperactivity theories will be categorized in a similar fashion to Moss and Garrett (1984). These authors divided muscle hyperactivity theories into theories that suggest a local cause and those that suggest a centrally mediated cause.

1.9.1.d(1)...Local Causes of Hyperactivity

The main local cause of hyperactivity was thought to be dental malocclusions (Moss and Garrett, 1984). Early investigators believed that problems with dental occlusion, such as centric prematurity, balancing and premature contacts, working interferences and loss of posterior teeth, were closely related to the occurrence of TMD (Kumai, 1993). Bakke and Moller (1980) found that significant asymmetry of muscle activity could be detected when premature contacts were evident. Yemm (1976) reviewed the literature on whether a malocclusion causes increased muscle activity. He reported that sensory input from the periodontal mechanoreceptors results in a reflex inhibition of the jaw closing muscles and activation of the jaw opening muscles. Thus, he concluded that there was no evidence of jaw elevator muscle hyperactivity due to premature contacts or malocclusion. Rugh *et al* (1984) tested the hypothesis that occlusal discrepancies elicit nocturnal bruxism. The investigators measured nocturnal bruxism before, during, and after a crown with a deflective occlusal contact was placed for 10 to 21 days. Nine of their 10 subjects showed a drop in their nocturnal masseter activity after placement of the crown. The investigators concluded that nocturnal bruxism was not elicited by experimental deflective occlusal contacts.

1.9.1.d(2)...Centrally Mediated Causes of Hyperactivity

Many investigators believe that psychic tension or emotional stress are closely related to abnormal muscle activity (Kumai, 1993). Other investigators proposed a vicious cycle theory of hyperactivity (Okeson, 1985).

1.9.1.d(2).i.... Psychological Theories

The two major centrally mediated theories are Schwartz's (1981) theory of "disregulation, disordered homeostasis, and disease" and Laskin's (1969) "psychophysiologic" theory. Cannistraci and Fitz (1983) describe the "disregulation" theory as a neuropsychological separation of the brain from the body so that feedback signals are altered or lost (Cannistraci and Fitz, 1983). The "disreguation" theory proposes that this loss of communication is exacerbated by stress and that it results in muscle hyperactivity and pain. The goal of treatment, such as biofeedback, is to renew appropriate contact between the brain and the masticatory muscles. This theory is supported by Glaros (1996), who found that TMD patients have deficits in proprioceptive awareness during periods of nonstress when compared to nonpain subjects.

Similar to the "disregulation" theory, the "psychophysiological" theory of hyperactivity predicts that psychological stress causes a centrally mediated hyperactivity response in the muscles of mastication (Laskin, 1969; Yemm 1969a, b, c; Rugh and Solberg, 1976). This theory has received support in studies where experimentally-induced stress has produced a significantly greater increase in masticatory muscle activity of TMD patients compared with controls (Yemm, 1969c; Mercuri *et al*, 1979; Katz *et al*, 1989).

Moss and Garrett (1984) criticized the studies by Yemm (1969c) and Mercuri *et al* (1976) for lacking a resting period between trials, a lack of control for EMG artifacts caused by performing the stressful tasks, and inadequate statistical analysis. Katz *et al* (1989) addressed some of these criticisms and found significant differences between the right temporalis muscle activity of TMD patients and control subjects during stressful stimuli. However, the relatively small difference in the right temporalis and the lack of statistically significant differences in the EMG activity of bilateral masseter and the left temporalis muscles caused the authors to question the clinical significance of their findings.

Studies have attempted to determine personality correlates of TMD patients. TMD patients with concomitant myogenous and arthrogenous symptoms reported more stress and stronger stress-related emotional reactions than patients with only a joint or muscle component to their TM disorder (de Leeuw *et al*, 1994). Patients with TMD experienced less personal control over their health and reported to cope differently with stress than controls (de Leeuw *et al*, 1994). However, Harkins *et al* (1991) did not find a significant relationship between personality and symptoms of TMD. Unfortunately, because the studies relating personality to TMD were primarily correlational, causal relationships were difficult to establish (Rugh and Solberg, 1976).

1.9.1.d(2).ii.... Vicious Cycle Theory

This theory (Okeson, 1985) stated that dysfunction causes pain which then reinforces the dysfunction. For example, the pain produced by myospasms of the muscles of mastication can self-perpetuate further myospasms because of the cyclic effect of pain on emotional stress. When muscle splinting or spasm continues uncontrolled, the pain of the muscle splinting feeds back to the muscle. The pain experienced with this spasm can increase the activity of the gamma efferents, which in turn increases muscle activity. The significance of this theory is that myospasms can continue even after the original cause of the myospasm is removed. This theory has been criticized because of the controversy surrounding the research that muscle hyperactivity is an etilogical factor of TMD (Lund and Widmer, 1989; Lund *et al*, 1991).

1.9.2...The Centrally Mediated Theory of TMD

Lund *et al* (1991) proposed an alternative pain-adaptation model to the theory that muscle hyperactivity is an etiology of TMD. They suggested that pain arising from joints, teeth, and other non-muscular tissues causes the same signs of dysfunction as muscle pain because the interneurons receive converging excitatory inputs from different tissue. They also proposed that any elevation of EMG activity is part of a general adaptive response to pain that includes a change in facial expression or head posture. For example, it has been found that patients with chronic lower back pain were scored with higher levels of pain behaviors such as grimacing or sighing (Keefe and Hill, 1985). Data from a study by Lund *et al* (1991) suggested that there is a decrease in motoneuron output when the masticatory muscle is acting as an agonist and an increase in output when is acting as an antagonist. They explained this adaptation as a protective mechanism and based their explanation for this mechanism on the pain-adaptation model. More research is required before this model can be accepted as an alternative theory to the hyperactivity theories.

1.10...Conclusion

Because the theories on the etiology of TMD lack strong support, it is impossible to advocate any single theory. The current classification system and variable use of nomenclature has made the establishment of an etiological theory more difficult. In addition, the relationship of the masticatory muscles to the TMJ adds to the confusion because problems with the muscles can occur secondarily to problems with the TMJ and vice versa (Moss and Garrett, 1984).

Thus, researchers have concluded that TMD is best viewed as the result of a number of interlocking factors of occlusal, neurophysiological, and psychological origin (Moss and Garrett, 1984; Okeson, 1996). It is important that further research is done to continue to develop the theories of the etiology of muscle pain in TMD patients and that dentists keep an open mind to these different concepts so that patients receive the best possible care.

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Chapter 2

Research Paper

Electromyographic Data and Post-exercise Pain in Female Muscle Pain and Control Subjects after Experimental Chewing

2.1...Introduction

Temporomandibular disorders (TMD) comprise a group of conditions characterized by pain and tenderness in the masticatory muscles and/or temporomandibular joint (TMJ) (Okeson, 1996). Some TMD subjects reported that chewing or mastication caused or aggravated their facial pain (Moss *et al*, 1984; Glass *et al*, 1993b). Self-reports of pain with chewing by TMD patients varied from approximately 37% (Zarb and Thompson, 1970; Fricton *et al*, 1985; Dworkin *et al*, 1990) to 68.4% (Bush *et al*, 1989). When TMD patients were selected specifically for having pain in the masticatory muscles, the self-reporting of pain with chewing varied from 67.2% (Dao *et al*, 1994) to 81% (Bush *et al*, 1989). Dao *et al* (1994) found that a controlled three minute wax-chewing test increased pain intensity in approximately half (48.3%) of a group of 60 TMD patients suffering from muscle pain. An unusual finding by these investigators was that many (31.7%) subjects experienced a decrease in their pain intensity after chewing. The remainder (20%) reported no change in pain following the chewing exercise.

One explanation for pain following a chewing exercise is found in the "muscle hyperactivity" theory, which proposes that hyperactivity of the muscles of mastication causes pain in TMD patients. This hyperactivity is thought to be due to a variety of causes, including muscle fatigue (Christensen, 1986; Bakke and Moller, 1992) and psychological stress ("psychophysiological" theory) (Laskin, 1969; Yemm 1969a,b,c; Rugh and Solberg, 1976; Mercuri *et al*, 1979; Katz *et al*, 1989). Some authors have criticized the "muscle hyperactivity" theory because of inadequate experimental evidence that hyperactivity is linked to TMD (Moss and Garrett, 1984; Lund and Widmer, 1989; Lund *et al*, 1991). Supporters of the "psychophysiological" theory have questioned the clinical significance of their findings because of small differences between the EMG activity of TMD and normal subjects (Katz *et al*, 1989). The "muscle hyperactivity" theory predicts that resting EMG activity is higher on the side of unilateral pain. However, no significant difference in EMG activity has been found between painful and non-painful sides (Majewski and Gale, 1984; Dahlstrom *et al*, 1984). In addition, the theory predicts that biofeedback therapy, which reduces EMG activity, will also reduce pain symptoms. However, no statistically significant relationship has been established between reduced EMG activity and pain symptoms (Peck and Kraft, 1977; Burdette and Gale, 1988).

The effect of a chewing exercise on baseline EMG levels is important to testing the "muscle hyperactivity" theory because of the association between mastication and pain. While there have been many studies of the EMG activity in jaw muscles during mastication in healthy patients (Wood, 1987; McCarroll *et al*, 1989; Balkhi *et al*, 1993; van Eijden *et al*, 1993; Blanksma and van Eijden, 1995; Christensen *et al*, 1996) and TMD patients (Stohler and Ash, 1986; Dolan and Keefe, 1988; Kumai, 1993), there have been very few studies of the EMG activity after chewing in these two groups. Tzakis *et al* (1994) measured muscle activity before and after a 30 minute gum-chewing exercise. Postural EMG activity and the activity during a maximum clench in the intercuspal position were recorded before and after chewing. They found no differences between the experimental pain and non-pain control groups. One criticism of this study is that there was no indication of the amount of time between the end of mastication and starting the EMG recordings. Second, the experimental group consisted of healthy subjects in whom pain was achieved through vigorous mastication. No patients with a history of muscle pain were included in their study.

The purpose of this study was to test the "muscle hyperactivity" theory by comparing the post-chewing postural EMG data between TMD patients with muscle pain and non-pain subjects. This theory predicts that TMD patients with muscle pain will have higher post-masticatory postural muscle activity than non-pain subjects. This prediction was tested by measuring the EMG muscle activity of the left and right masseter and anterior temporalis muscles directly following a chewing exercise in a group of female TMD patients with muscle pain and a non-pain group of females. The second purpose of this study was to observe relationship between post-masticatory EMG levels and post-masticatory pain scores in pain subjects. The "muscle hyperactiviy" theory predicts that there is a correlation between EMG activity and the ipsilateral pain symptoms (Lund and Widmer, 1989).

Only female subjects between the ages of 18 and 40 were chosen because females reported more frequently for treatment of TMD than males by a ratio of approximately 3:1 (Rugh and Solberg, 1985) and because gender-related differences in EMG recordings have been observed (Visser and De Rijke, 1974). In addition, the age group of the subjects was controlled because age has been found to affect EMG activity (Carlsson, Alston, and Feldman, 1964). The masseter and anterior temporalis muscles were studied because EMG activity from these sites is commonly measured in biofeedback treatment, which has been used to treat TMD patients with muscle pain (Glass *et al*, 1993a).

2.2...Materials and Methods

Subjects

The sample population included 27 female muscle pain subjects with a mean age of 25.3 years (range of 19 to 39 years) and a control group of 20 non-pain female subjects with a mean age of 23.7 years (range of 19 to 33 years). An independent samples *t*-test showed that the mean ages of the experimental and control groups were not statistically significantly different. The pain subjects were recruited through announcements published in local newspapers and posted on the University of Alberta campus. They were also chosen from patients attending the University of Alberta TMD Clinic. The controls were recruited from university students and employees.

The selection criteria for the muscle pain patients were similar to that of Dao *et al* (1994) with certain modifications. Subjects were required to be females between ages 18 and 40 and to have experienced facial pain at least two times per week for the duration of at least two of the previous months. An additional criterion was tenderness to palpation in at least three sites in the muscles of mastication. Exclusion criteria included the use of muscle relaxants and/or antidepressants, the detection of muscular trigger points, and a history of head and neck trauma, such as a car accident, occurring less than one year ago.

Following a previous protocol (Reid *et al*, 1994), any subjects wearing a splint or nightguard left them out for a minimum of two weeks prior to the initial EMG recording session. Eight pain subjects were regular splint wearers. Although the presence of a TMJ click was not an exclusion criterion, the purpose of the screening examination was to identify and exclude patients with intra-articular TMJ pathosis such as any of the arthritides.

Non-pain subjects were subject to the same exclusion criteria. Subjects were not included in the control group if they had any history of facial pain or if they reported tenderness to palpation.

Procedure

All subjects completed a questionnaire, underwent a clinical examination by a TMJ clinician different than the principal investigator, and gave informed consent to procedures approved by the Research Ethics Board of the University of Alberta Faculty of Medicine and Oral Health Sciences. Each subject came to two sessions for the measurement of electromyographic (EMG) muscle activity before, during, and after a brief chewing exercise. The principal investigator did not know the categorization of each subject. Visits were made at least 1 day apart and no more than 1 week apart. With some exceptions, the second session was booked at the same time of day as that of the first session. A brief description of the experiment and instructions were read to each subject to ensure that all participants were given the same information.

The subject's skin over the belly of the masseter and anterior temporalis muscles was cleaned vigorously with a cotton gauze (Johnson and Johnson Medical Inc., Arlington, Texas, USA) and facial cleanser (Clearasil Deep Cleanser-Sensitive, Proctor and Gamble, Inc., Toronto, Ontario, Canada) prior to application of the electrodes. Four sets of two bipolar silver-silver chloride surface electrodes (SensorMedics Corp., Yorba Linda, California, USA) were placed bilaterally to measure the EMG muscle activity of the right and left masseter and anterior temporalis muscles. The 9 mm diameter electrodes were filled with electrode paste (Teca-Vickers Medical and Electrode Electrolyte, Teca Corp., Pleasantville, New York, USA.). Two electrodes were placed approximately 15 mm apart over the bulk of each muscle, parallel to the direction of the muscle fibres. For the anterior temporalis muscle, the electrodes were placed level with the eyebrow, as close to the hairline as possible. A ground electrode was placed on the skin proximal to the spinous process of the seventh cervical vertebra. The inter-electrode impedence was measured with a voltmeter immediately after electrode placement to ensure that the resistance was less than 10KΩ for each muscle. If the resistance was greater than this value, the electrode was removed, cleaned, and replaced. The resistance was rechecked at the end of the experiment in order to ensure that the electrodes were correctly positioned for the duration of the experiment.

The 4-channel EMG signals were amplified by preamplifiers (P15, Grass Medical Instruments, Quincy, USA). The gain was set at 1000 and the filter bandwidth at 100-Hz – 1000-Hz. The raw signals were displayed on an 8-channel oscilloscope (5A14N, Tektronix, Beaverton, U.S.A.) and fed through a 12-bit analog/digital converter at 1000-Hz/channel (Dash-8, Mytrabytes, Ltd., Wilminton, U.S.A.). The digitized data were picked up by a data acquisition board (Dash-8) installed in a personal computer (IBM compatible 486/33 MHz with math coprocessor) and were recorded in 20 second samples. One pre-chewing and three post-chewing recordings (immediately post-chewing, 1 minute post-chewing, and 3 minutes post-chewing) were recorded. After the data was smoothed and rectified using moving averages, each 20 second sample was subdivided into quartiles. The average EMG activity over a 2-second interval at the start of each quartile was calculated. Thus, for each 20 second sample, four average EMG measurements were calculated at the following time intervals: 0-1 second, 5-6 seconds, 10-11 seconds, and 15-16 seconds.

After the electrodes had been placed and before the recording was started, subjects were asked to rate their pain separately for the right and left sides on a 100 mm visual analog scale (VAS) where 0 represented no pain and 100 represented intense pain. Subjects were seated in an upright position on a dental chair with their legs uncrossed and their heads supported by a headrest. To minimize EMG artifact, they were instructed to assume a comfortable posture with no extraneous movement or talking while data were collected. They were also instructed to keep their lips closed and their teeth slightly apart during the resting portions of the study. This was to avoid the possibility of bruxing or clenching. A calm, quiet atmosphere was provided. All subjects listened to Pachabel's Canon in D for the duration of the experiment.

The total length for each recording session was approximately 17 minutes. Following 8 minutes of resting baseline, each subject was asked to chew half a leaf of green casting wax, gauge 28 (Kerr, California, USA) for four minutes. The subjects were allowed to chew at their rate of preference and side of preference except for four 20 second intervals when unilateral chewing EMG samples were recorded (twice on the right and twice on the left) for analysis in a further study. After the exercise, subjects were asked to keep the wax in their mouth for 20 seconds with their lips closed and teeth slightly apart while a resting EMG record was made. The purpose of this was to prevent EMG artifact from the movement required to remove the wax from the mouth. The wax was discarded for the subsequent 2 recordings during the 5 minute post-exercise EMG recording. After the final resting record, patients again assessed their level of pain.

2.3 Statistics

The EMG data for sessions 1 and 2 were not pooled because a paired *t*-test showed that there was a statistically significant difference between sessions in the EMG data collected. Instead, the following statistical tests were applied separately to each session. Because including graphical results of both sessions would be repetitive, only session 2 data was presented in the results. Due to missing data, EMG data from two pain subjects and pain data from one pain subject were excluded from the following statistical tests. All statistical tests were reported at a probability level of 0.05.

EMG

First, an independent-samples *t*-test was used to compare the mean pre-chewing and post-chewing postural EMG activity between the pain and non-pain groups for each muscle. Second, the difference between post- and pre-chewing postural EMG data was calculated for each muscle of each subject. This was referred to as the "relative change" in muscle activity. A second independent-samples *t*-test was used to compare the mean "relative change" in EMG activity between the pain and non-pain groups for each muscle.

Pain Ratings and EMG

First, the pain subjects were divided into those who had increased pain after chewing, those who had no change, and those who had decreased pain (Table 1; p. 79). This was done to compare the post-chewing pain to the results of Dao *et al* (1994). An increase or decrease in pain score was defined as a change in VAS of 5.0 mm or greater between the pre- and post-chewing score (Dao *et al*, 1994). Second, a linear regression analysis was used to assess the relationship between the pre- and post-chewing EMG activity and the ipsilateral pre- and post-chewing pain scores. For example, left side post-chewing EMG activity was compared to left side post-chewing pain score. The dependent variable was pain and the independent variable was EMG activity.

2.4...Results

EMG

There was little difference between the mean postural pre-chewing EMG activity of the pain and non-pain subjects (Figures 2-1, 2-5, 2-9, 2-13; pp. 73-7). Only the muscle activity of the right masseter (Figure 2-5; p. 74) of the pain subjects was statistically significantly ($p \leq 0.05$) elevated in the pre-chewing sample relative to the non-pain subjects.

The mean postural EMG activity in all muscles of both the pain and non-pain subjects immediately post-chewing was invariably greater than the pre-chewing activity. (Figures 2-2, 2-6, 2-10, 2-14, 2-17 to 2-20; pp. 73-8). This [illegible]se was greater in the pain than non-pain subjects. However, independent-samples *t*-tests indicated that these differences between the samples were statistically significant ($p \leq 0.01$) only in the left masseter (Fig. 2-2; p. 73) during the first 20 seconds after the chewing exercise. Muscle activity of the right temporalis of the pain subjects was increased relative to the non-pain subjects ($p \leq 0.05$; Figs. 2-14, 2-15; p.76) during only a portion of the time samples recorded immediately after and one minute after the chewing exercise.

A similar difference between the pain and non-pain groups appeared when the mean "relative change" in immediately post-chewing EMG activity was compared between the two groups (Figures 2-17 to 2-20; pp. 77-8). All subjects' muscles had increased activity in the immediately post-chewing sample but the differences were greatest in the left masseter and right temporalis muscles. In addition, the differences were consistently larger in pain than non-pain subjects. However, an independent-samples *t*-test determined that only the left masseter muscle showed a statistically significant ($p \leq 0.05$; Fig 2-17; p. 77) difference between both groups.

Pain Ratings and EMG

All non-pain (n = 20) patients reported no pain prior to or following the chewing exercise. In contrast, all pain patients (n = 26), except two, reported pain before and after the chewing exercise. The categorization of pain subjects according to their pain responses is summarized in Table 2-1 (p. 79). After exercise, pain scores increased on the left side in 42.3% (n = 11) of the pain subjects and on the right in 57.7% (n = 15) of the pain subjects. No change in pain was found on the left side of 42.3% (n = 11) of the pain subjects and on the right side in 38.5% (n = 10) of the pain subjects. Only 15.4% (n = 4) of the pain subjects had a decrease in pain on the left and 3.8% (n= 1) had a decrease on the right.

A linear regression analysis (Table 2-2; p. 80) was done using only pain subjects' EMG pre- and post-chewing EMG activity and ipsilateral pre- and post-chewing pain scores. The R^2 values calculated in the regression analysis were low, ranging from 0 to 0.318, indicating that the variability in the pain scores could only partially be explained by the variability in the EMG activity.

2.5...Discussion

The EMG data for sessions 1 and 2 were not pooled because a paired *t*-test showed that there was a statistically significant difference in EMG data collected during sessions 1 and 2. Uncontrolled variables, such as electrode placement and the subjects' emotional and/or physical states, may have contributed to this discrepancy. The statistical tests were done for sessions 1 and 2 but only the results from session 2 were reported to avoid repetition. There was no statistically significant difference between the EMG activity of the pain and non-pain subjects during session one, which differed slightly from the results of session 2. Session 2 data was chosen for reporting because research has shown that an "orienting response" can affect a subject's initial EMG response to an experimental protocol (Grossman, 1973). The reactive effects of the "orienting response" can sometimes be identified by initially high or low EMG readings that usually stabilize during subsequent monitoring (Van Toller, 1979; Hudzinski and Walters, 1987).

Some previous studies have shown that there was a difference in resting EMG activity between TMD patients and non-pain subjects (Lous *et al*, 1970; Dohrmann and Laskin, 1978; Rugh and Montgomery, 1987). Other investigators have not found a difference in postural EMG between pain and non-pain subjects (Majewski and Gale, 1984; Katz *et al*, 1989). This study showed that the pre-chewing resting baseline muscle activity was not increased in the pain subjects in all four muscles. The only muscle in which pain subjects' pre-chewing EMG activity was increased relative to the non-pain subjects was the right masseter. The reason for this finding in the right masseter is unclear. Research has shown that such differences in postural EMG activity could be related to minor changes in mandibular posture (Katz *et al*, 1989). Second, it is possible that the surface electrodes applied to the anterior temporalis region, through volume conduction, may have recorded activity of the muscles of facial expression (Katz *et al*, 1989).

The "muscle hyperactivity" theory predicts that EMG activity would be elevated after a chewing exercise, when a pain subjects experience pain in their muscles of mastication. In this study, none of the non-pain subjects reported any pain before or after chewing. However, all pain subjects, except two, reported pre- and post-chewing pain. The mean EMG activity in pain subjects was increased relative to that of non-pain subjects in only the left masseter during the 20 seconds immediately post-chewing. When each subject's pre-chewing EMG activity was subtracted from her post-chewing data to determine the magnitude of the increase in each subject's "relative change" in muscle activity following a chewing exercise, a similar result emerged. Only the left masseter muscle showed a statistically significantly greater immediately post-chewing increase in pain subjects. However, this increased EMG activity lasted for a short duration, occurring only in the first 20 second recording after the chewing exercise. The clinical significance of this finding is questionable since it did not take pain subjects more than one minute to return to their baseline EMG activity compared with the non-pain controls. Also, out of four muscles that were monitored, only the left masseter exhibited an increased activity in pain subjects relative to those without pain. Thus, the results provide little support for the "muscle hyperactivity" theory but they do provide support for the findings of Tzakis *et al* (1994), who found no significant difference in the EMG postural activity between their experimental and control groups after a thirty minute chewing exercise.

The reason for this difference in left masseter EMG activity between pain and non-pain subjects is unclear. This study does not support the hypothesis that the increase in activity is related to pain. For example, the linear regression analysis did not indicate a stronger relationship between left masseter EMG data and left side pre- and post-chewing pain scores than the other muscles. In addition, a larger proportion of subjects reported an increase in pain following the post-chewing exercise on their right side (57.7%) than on their left side (42.3%). Previous studies which have compared resting EMG activity on the side of unilateral pain to EMG activity on the non-pain side have shown no statistically significant difference in EMG levels between the sides (Majewski and Gale, 1984; Dahlstrom *et al*, 1985).

A limitation of this study was that 20 second samples were recorded instead of a continuous measurement of the EMG activity because of inadequate storage capability of the computer system. Thus, some valuable information between samples was lost. For example, immediately after chewing, there was a 40 second delay between the end of the first recording and the start of the second recording. This delay was essential for the principal investigator to prepare the computer for the second recording. Ideally, the entire 17 minute recording session would have been continuously recorded so that all fluctuations in EMG activity, especially following chewing, could have been measured.

Some of the pain response findings in this study differ from that by Dao *et al* (1994). In their study, 48.3% of the pain subjects reported an increase in VAS pain intensity of more than 5 mm. This is comparable to our left (42.3%) and right (57.7%) pain scores. However, Dao *et al* (1994) reported that 31.7% of their subjects reported a decrease in pain while very few subjects in this study (15.4% on the left and 3.8% on the right) reported such a decrease. Finally, Dao *et al* reported that 20.0% of their pain subjects reported no change in pain. In this study, the percentage of subjects reporting no pain was increased relative to Dao *et al*'s sample (42.3% on the left and 38.5% on the right).

Dao *et al* (1994) suggested that opposing reactions to chewing exercise (an increase versus a decrease in pain) could indicate two distinct pathologies. This study does not provide support for the hypothesis that two pathologies exist because of the much smaller percentage of subjects with a decrease in pain (15.4 % on the left and 3.8% on the right) in this study. The same material was chewed in both studies and the chewing time in this study was 4 minutes was similar to the 3 minute exercise in the study by Dao *et al* (1994). It is possible that 1 additional minute of chewing could have resulted in the difference in results between the studies. Further research should be done to determine if different pain responses occur with changes in chewing times.

The results of the linear regression analysis do not support the hypothesis that there is a relationship between EMG activity and pain levels of pain subjects because of the low R^2 values, which ranged from 0 to 0.318. These low values indicated that the variability in EMG recordings was only partially due to the variability in pain scores. The findings from the regression analysis are not surprising. All, except two, of the pain subjects reported pain before and after chewing and a significant percentage of the pain subjects reported an increase in pain after chewing compared to no reported pre- and post-chewing in the non-pain subjects. However, there were no statistically significant differences, except in the left masseter, between the EMG activity of pain and non-pain groups. This finding is supported by previous studies which have not found a reliable relationship between pain scores and EMG activity (Peck and Kraft, 1977; Dahlstrom *et al*, 1984; Burdette and Gale, 1988).

The cause of increased pain during and after chewing is speculative. Some studies suggested that pain experienced by TMD patients during and after chewing was related to simultaneous abnormal electromyographic (EMG) muscle activity. Kumai (1993) used EMG data to demonstrate that TMD subjects with unilateral pain symptoms chewed gum in an asymmetric pattern when compared to subjects with no pain but he did not find a reliable relationship between the reported side of dysfunction and the side showing an abnormal chewing pattern. Stohler and Ash (1986) reported prolonged contraction times and greater EMG amplitudes for chewing cycles associated with pain compared with those free of pain. Dolan and Keefe (1988) found that "high" pain right-sided TMD subjects had higher activity in the left masseter than the right when chewing gum on their right side. This was contrary to the finding that the masseter muscle activity was usually higher on the side on which one was chewing (Moore, 1993; Christensen *et al*, 1996). It is possible that an imbalance in EMG activity during chewing, such as that found by Dolan and Keefe (1988), might have contributed to the brief differences in post-chewing postural EMG activity recorded in the left masseter in this study. Analysis of the EMG data during mastication would have helped to determine if this increased EMG activity in the left masseter of pain subjects could be related to intensity of EMG activity and rate of chewing. Future analysis of this data is planned.

This study could have been strengthened by narrowing the criteria for the selection of subjects in both the muscle pain and non-pain group. For example, bruxers were included in both the pain and non-pain groups. Sherman (1985) found that postural EMG activity was elevated in bruxers, whether or not they suffered from facial pain. However, Sherman's study could be critiqued for his unclear description of how he determined that subjects were assessed for bruxism. He only indicates that they were included in a bruxism group if there was "physical evidence" of a history of clenching and bruxism. Today, bruxism habits are best assessed by portable electromyography and sleep laboratories because other methods of assessment of bruxism have been shown to be unreliable (Okeson, 1996). In the future, it might be useful to use portable EMG to screen for bruxism subjects when doing studies on postural EMG activity.

A confounding variable in this study was the relaxing music played during EMG recording session. This was done in an effort was made to reduce the amount of stress for the subjects during the EMG recordings. However, Moller *et al* (1971) found that relaxation reduced the enhanced postural activity in pain patients. The "psychophysiological" theory states that psychological stress initiates the muscle hyperactivity via a centrally-mediated response (Katz *et al*, 1989). There is experimental evidence of increased EMG activity in pain subjects compared with non-pain subjects during stressful stimuli (Yemm, 1969c; Mercuri *et al*, 1979; Dolan and Keefe, 1988; Katz *et al*, 1989). The encouragement of relaxation may have influenced the postural EMG activity of the subjects.

In fatigue studies, Hellsing and Lindstrom (1983) concluded that all members of a synergistic group of muscles should be simultaneously monitored because a reduced activity in one muscle may be compensated by an increased activity in another. The same may be true of studies that look at resting baseline activity. For example, medial pterygoid activity has been rarely studied because needle electrodes are required to measure its activity. The posterior temporalis is not often included in EMG studies for the same reason. It is possible that recording the synergistic activity of all jaw elevator muscles could give investigators a clearer picture of the postural activity before and after a chewing exercise.

2.6...Conclusions

Although the mean EMG activity in the left masseter muscle of pain patients was elevated in comparison to non-pain subjects, the magnitude of elevation was small, it was not observed in all muscles, and the duration of this elevated muscle activity was low. Thus, pain and non-pain female subjects show a similar recovery to postural EMG levels following a four minute wax-chewing exercise. Linear regression analysis showed that very little of the variability in EMG activity was due to the variability in pain scores. Thus, it was concluded that there was no statistically significant relationship between EMG activity and the ipsilateral pain scores following a chewing exercise. This study does not support the "hyperactivity" theory as an etiology of TMD.

A version of this chapter has been submitted to the Journal of Orofacial Pain.

Figures of Mean Left Masseter EMG Activity vs Time (Pain and Non-pain)

Fig. 2-1: Left Masseter Prior to Chewing

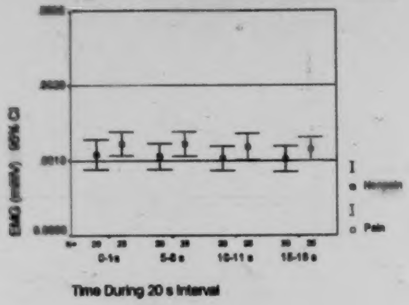
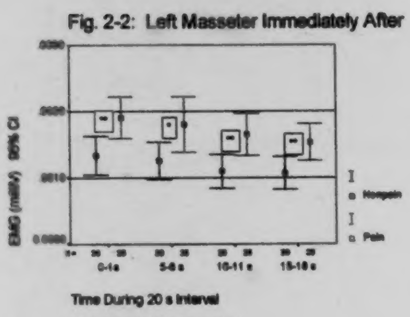
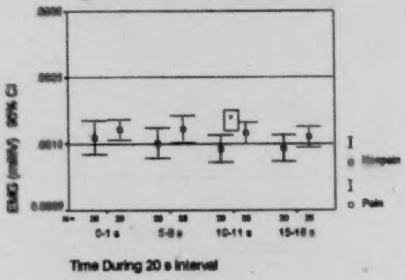


Fig. 2-2: Left Masseter Immediately After



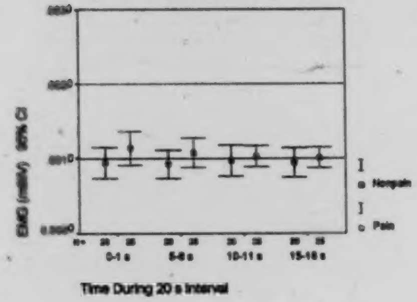
* = p-value < 0.05; ** = p-value < 0.01

Fig. 2-3: Left Masseter 1 Min After



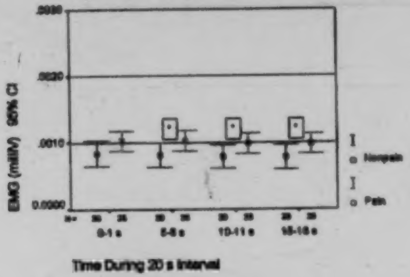
* = p-value < 0.05

Fig. 2-4: Left Masseter 3 Min After



Figures of Mean Right Masseter EMG Activity vs Time (Pain and Non-pain)

Fig. 2-5: Right Mass. Prior to Chewing



* = p-value < 0.05

Fig. 2-6: Right Mass. Immediately After

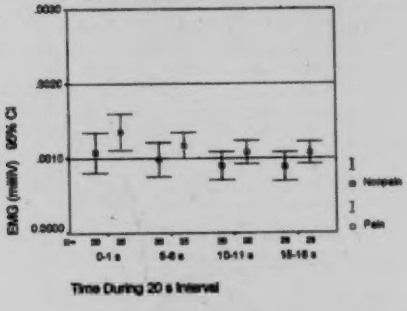


Fig. 2-7: Right Masseter 1 Min After

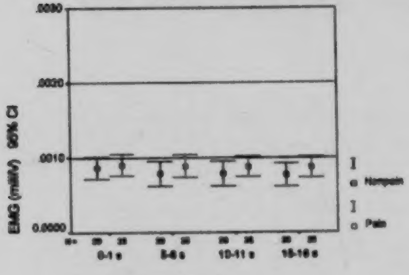
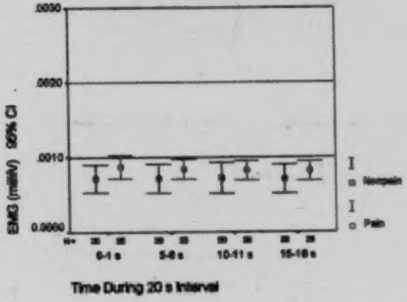
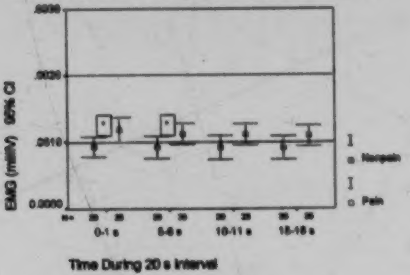


Fig. 2-8: Right Masseter 3 Min After



Figures of Mean Left Temporalis EMG Activity vs Time (Pain and Non-pain)

Fig. 2-9: Left Temp. Prior to Chewing



* = p-value < 0.05

Fig. 2-10: Left Temp. Immediately After

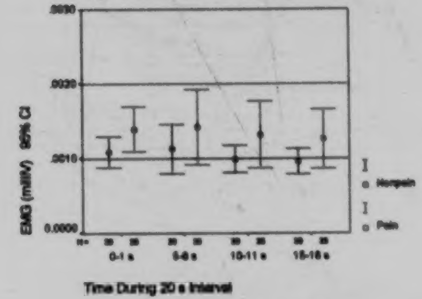
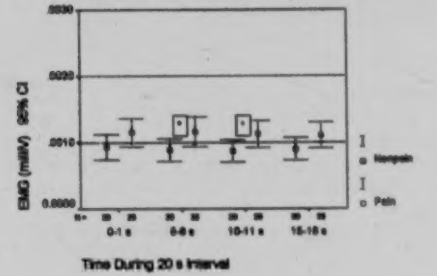
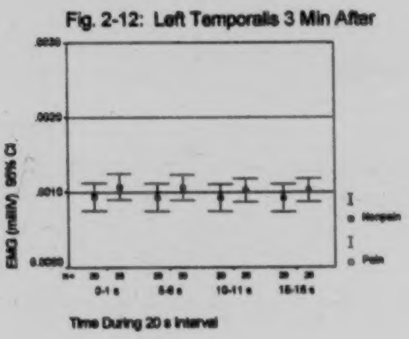


Fig. 2-11: Left Temporalis 1 Min After



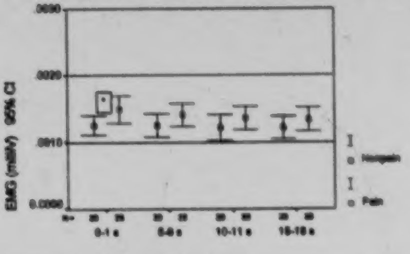
* = p-value < 0.05

Fig. 2-12: Left Temporalis 3 Min After



Figures of Mean Right Temporalis EMG Activity vs Time (Pain and Non-pain)

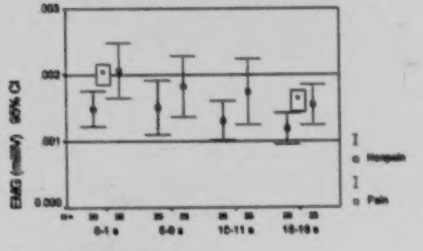
Fig. 2-13: Right Temp. Prior to Chewing



Time During 20 s Interval

* = p-value < 0.05

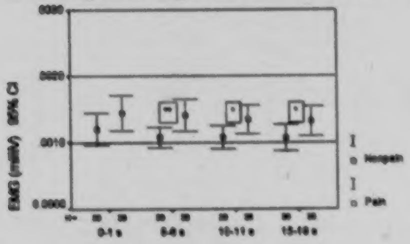
Fig. 2-14: Right Temp. Immediately After



Time During 20 s Interval

* = p-value < 0.05

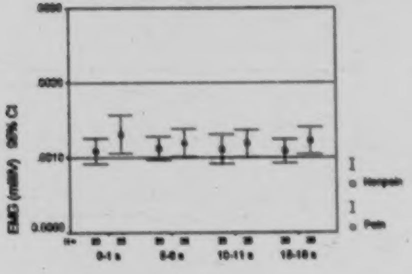
Fig. 2-15: Right Temporalis 1 Min After



Time During 20 s Interval

* = p-value < 0.05; ** = p-value < 0.01

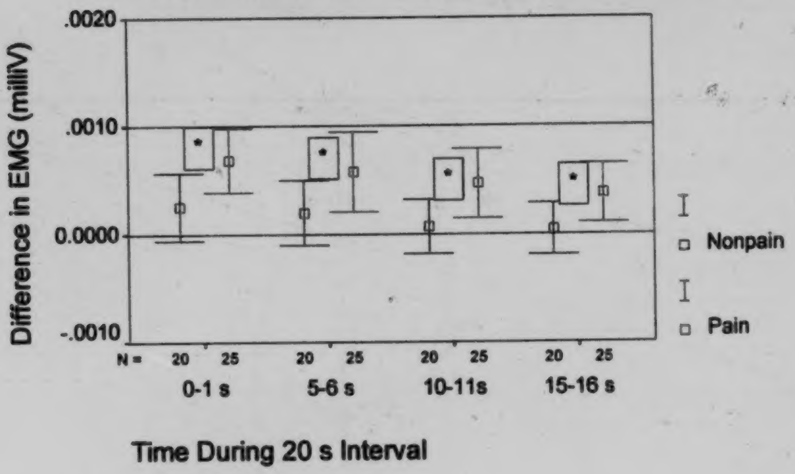
Fig. 2-16: Right Temporalis 3 Min After



Time During 20 s Interval

Difference Between Immediate Post-chewing EMG and Baseline EMG in the Masseter Muscles vs Time (Pain and Non-pain)

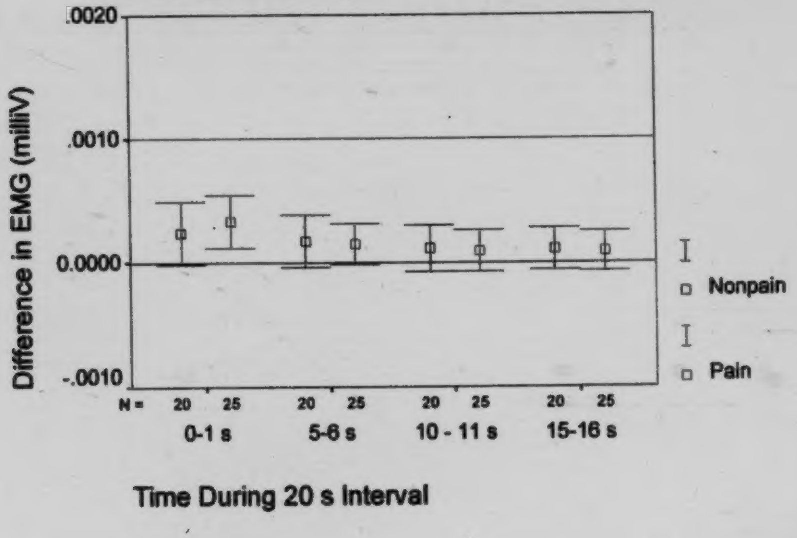
Figure 2-17



Left Masseter: Immed After - Baseline

* = p-value < 0.05

Figure 2-18



Right Masseter: Immed After - Baseline

Difference Between Immediate Post-chewing EMG and Baseline EMG in the Temporalis Muscles vs Time (Pain and Non-pain)

Figure 2-19

Left Temporalis: Immed After - Baseline

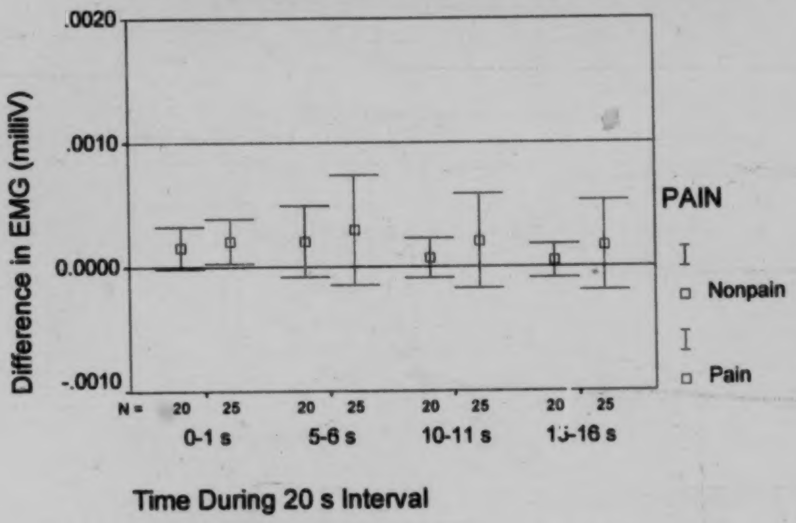


Figure 2-20

Right Temporalis: Immed After - Baselin

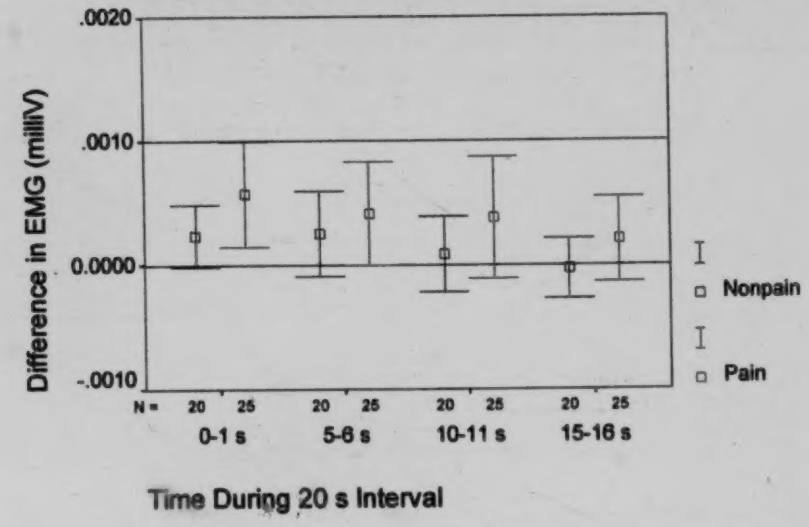


Table 2-1. VAS Data: Changes in Pain Intensity After Exercise

	% of Patients (n)	
	Left	Right
Increase[a]	42.3% (11)	57.7% (15)
Decrease[a]	15.4% (4)	3.8% (1)
No Change[b]	42.3% (11)	38.5% (10)
Total	100% (26[c])	100% (26[c])

[a] An increase (or decrease) was defined as a change in VAS pain intensity > 5 mm

[b] A change < 5 mm

[c] One missing value

Table 2-2. Results of Linear Regression on EMG and Pain Scores in Muscle Pain Subjects

Muscle	Time Period	R^2	Coefficient	P-Value
Left Masseter	Prior to	0.256	0.513	<0.001***
	Immediately After	0.168	0.420	<0.001***
	1 Min After	0.065	0.272	0.006**
	3 Min After	0.009	0.096	0.344
Right Masseter	Prior to	0.001	-0.028	0.782
	Immediately After	0.020	0.174	0.084
	1 Min After	0.000	0.016	0.872
	3 Min After	0.036	0.214	0.032*
Left Temporalis	Prior to	0.204	0.460	<0.001***
	Immediately After	0.318	0.570	<0.001***
	1 Min After	0.279	0.535	<0.001***
	3 Min After	0.306	0.560	<0.001***
Right Temporalis	Prior to	0.003	-0.051	0.617
	Immediately After	0.042	0.227	0.023*
	1 Min After	0.001	0.104	0.302
	3 Min After	0.068	0.279	0.005**

* = $P \leq 0.05$; ** = $P \leq 0.01$; *** = $P \leq 0.001$

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Chapter 3

Discussion and Recommendations

3.1...General Discussion

Many patients present to their dentists and medical doctors complaining of symptoms involving their temporomandibular joint. One significant symptom of this disorder is masticatory muscle pain, which may or may not be associated with other symptoms. This pain may be treated in a variety of ways but no single treatment modality is appropriate to every patient.

The purpose of EMG biofeedback-assisted relaxation is to detect muscle activity and to display the electrical activity of a given muscle in the form of either audio or visual signals. The underlying principle of biofeedback is that lowering postural tonicity of muscles is desirable (Jankelson, 1990). The patient is made aware of his muscle activity by receiving the signal and learns how to control the firing of his muscle motor units by altering the signal (dos Santos, Jr., 1995). Many studies have shown improvement in TMD patients' symptoms using biofeedback treatment (Gessel, 1975; Carlsson and Gale, 1977; Dohrmann and Laskin, 1978; Gale, 1979; Flor and Birbaumer, 1993; Turk *et al*, 1993).

The mechanism by which biofeedback works is poorly understood and one hypothesis is that biofeedback helps patients develop relaxation skills and coping strategies (Gevirtz *et al*, 1995). A second hypothesis is that biofeedback helps subjects to reduce postural EMG activity, which then reduces pain (Gevirtz *et al*, 1995). This hypothesis of the mechanism of biofeedback is related to the "muscle hyperactivity" theory, which predicts that hyperactivity in the masticatory muscles causes pain in TMD patients. This theory has been supported by studies that showed higher postural EMG activity in TMD patients than non-pain subjects (Lous *et al*, 1970; Dohrmann and Laskin, 1978;

Rugh and Montgomery, 1987). However, the "hyperactivity" theory has been refuted in biofeedback studies that found a poor correlation between the decrease in EMG activity and the decrease in pain symptoms (Peck and Kraft, 1977; Dahlstrom *et al*, 1984; Burdette and Gale, 1988).

The effect of a chewing exercise on baseline EMG levels was important to test the "muscle hyperactivity" theory because of the association between mastication and pain. Dao *et al* (1994) found that a three minute wax-chewing test increased pain intensity in approximately half of a sample of MPD patients. The literature did not yield much research on the measurement of post-exercise EMG activity of the muscles of mastication. Tzakis *et al* (1994) measured the EMG activity of the right posterior temporalis, right anterior temporalis, and right masseter muscle activity before and after two 30 minute chewing fatigue tests. Their experimental group consisted of healthy males (N = 7) and females (N = 10) who chewed hard gum 1 hour daily for 28 days after the first and before the second fatigue test. The control group, also consisting of healthy males (N = 4) and females (N = 4), did not undergo a fatigue test or perform any chewing training. The investigators found no difference in EMG activity between their pain and control groups even though there was a difference in pain symptoms. Their study was criticized because the researchers did not indicate the amount of time that elapsed between the cessation of mastication and the EMG recordings. Second, the experimental group consisted of only healthy subjects in whom pain was achieved through vigorous mastication. Inducing pain in a healthy subject may not be a good model for a pain subject (Clark *et al* 1989, 1991).

This observational study evaluated the "muscle hyperactivity" theory in two ways. First, the baseline EMG masticatory muscle activity directly following a chewing exercise was compared between a group of female TMD patients with muscle pain and a female non-pain sample. Second, the relationship between post-masticatory EMG activity and post-masticatory pain was assessed. The results of this study are discussed below.

Previous studies have shown that there was a difference in resting EMG activity between TMD patients and non-pain subjects (Lous *et al*, 1970; Dohrmann and Laskin, 1978; Rugh and Montgomery, 1987). This study does not support those studies because the pre-chewing resting baseline muscle activity was not consistently increased in the pain groups across both sessions, similar to the findings of other investigators (Majewski and Gale, 1984; Katz *et al*, 1989). The differences that were found were small and could have been due to minor changes in mandibular posture (Katz *et al*, 1989). Another possibility is that the surface electrodes applied to the anterior temporalis region, through volume conduction, may have recorded activity of the muscles of facial expression (Katz *et al*, 1989). For example, the anterior temporalis muscle electrodes may have picked up signals from muscles surrounding the eye.

The "hyperactivity" theory predicts that EMG activity would be elevated after a chewing exercise in muscle pain subjects experiencing pain in their muscles of mastication. In the present study, the mean EMG data immediately following the four minute chewing exercise was found to be elevated above the resting EMG levels in both pain and non-pain subjects. However, the post-chewing data were not consistently different between the groups over both sessions.

There were some minor differences between session 1 and 2 recordings. In the first session, the difference between the mean EMG activity between the two groups was statistically significant only in the right temporalis for the first two seconds following chewing. In the second session, the difference in means was statistically significant in the left masseter for 20 seconds after the chewing exercise and in the right temporalis for the first one minute and 20 seconds after the chewing exercise.

A limitation of this study was that 20 second samples were recorded instead of a continuous measurement of the EMG activity because of inadequate storage ability of the computer system. Thus, some valuable information between samples was lost. For example, immediately after chewing, there was a 40 second delay between the end of the first recording and the start of the second recording. This delay was essential for the principal investigator to prepare the computer for the second recording. Ideally, the entire 17 minute recording session would have been recorded so that fluctuations in EMG activity, especially following chewing, could have been more accurately measured.

There are many variables that may have accounted for this difference between sessions. First, variability in positioning of the electrodes may have led to differences in data between sessions. This variable could have been better controlled by constructing an electrode template for each subject to facilitate better replication of the positioning of the electrodes during the second session. However, the principal investigator was the only person to position the electrodes, which helped to standardize electrode position. Second, variability in the physical and emotional state of the subjects might have caused differences between the sessions. It has been shown that psychological stress increases postural EMG activity (Yemm, 1969ab) and that this effect was more pronounced in TMD subjects (Yemm 1969c; Mercuri *et al*, 1979; Katz *et al*, 1989). Third, variability in the time of day that the EMG data were recorded could have resulted in differences between recording sessions since some patients were unable to return for their second recording at the same time as their first session. Finally, the "Hawthorne" effect may have led to different results between the sessions. For example, the subjects' familiarity with the procedure during session 2 might have given them a more relaxed state of mind, resulting in different EMG measurements than session 1.

When comparing sessions 1 and 2, similar patterns in muscle activity appear. In both the left masseter and right temporalis, greater increases of immediately post-chewing muscle activity relative to baseline were apparent in both experimental and control groups across both sessions. Meanwhile, the right masseter and left temporalis EMG recordings were not as elevated after the chewing exercise. Certain factors could have affected the symmetry of muscle activity. First, facial morphology might have had an impact on the results. For example, the masseter muscle with increased cross-sectional area had greater EMG activity when comparing left- to right-side EMG recordings in healthy patients (Naeije, McCarroll, and Weijs, 1989). Second, occlusal interferences were shown to result in masseter muscle asymmetry (McCarroll *et al*, 1989b). The presence of a centric-relation (CR) to centric-occlusion (CO) shift was not an exclusion criterion in this study because of the prevalence of CR-CO shifts in TMD patients (Posselt, 1952; Kumai, 1993). A difference in the positioning of electrodes between the right and left sides may have led to differences between the muscles. However, because these differences were consistent between both sessions, this is unlikely to have an influence on the results.

When each subject's pre-chewing EMG activity was subtracted from her post-chewing data to determine the magnitude of the increase in her muscle activity following a chewing exercise, a similar emerged. Only the pain subjects exhibited a statistically significantly increased mean EMG activity relative to the non-pain subjects in their left masseter muscle immediately after a chewing exercise during both recording sessions. Because this increased relative EMG activity was of short duration, being approximately 6 seconds in the first session and 20 seconds in the second session, the clinical significance of this finding is questionable. Also, of four muscles that were monitored, only the left masseter showed an increased mean relative EMG activity in the pain subjects.

The reason for this increased mean "relative" left masseter activity between pain and non-pain subjects is unclear. This study does not support the hypothesis that the increase in activity is related to pain. For example, the linear regression analysis did not indicate a stronger relationship between left masseter EMG data and left side pre- and post-chewing pain scores than the other muscles. In addition, a larger percentage of subjects reported an increase in pain following the post-chewing exercise on their right side than on their left side (Right/session 1: 59.3% vs. Left/session 1: 44.4% and Right/session 2: 57.7% vs. Left/session 2: 42.3%). Previous studies which have compared resting EMG activity on the side of unilateral pain to EMG activity on the non-pain side have shown no significant difference in EMG levels between the sides (Majewski and Gale, 1984; Dahlstrom *et al*, 1985). Analysis of the EMG data during mastication would be useful to determine if the increased relative left masseter activity immediately post-chewing could be related to activity during mastication. Dolan and Keefe (1988) observed that all patients with MPD showed statistically significantly higher left than right masseter EMG activity when asked to chew gum on the left-side. This was not observed in the right masseter when subjects chewed on their right. They had no explanation for these differences observed in the chewing data.

Some EMG researchers express EMG data as percentage EMG by dividing the absolute EMG data by the EMG data collected during maximum voluntary occlusal force. By normalizing the data using this method, it is possible that the results would have been different. For example, if normalization had been used and if the EMG data during MVOF had been higher in the left masseter than the other muscles, then the increased relative left masseter EMG activity would not have been significant. Although MVOF was measured in all subjects of this study, data in this study were not normalized because research has shown that pain subjects have a lower maximum output (Sheiholeslam *et al*, 1982; van Boxtel *et al*, 1983) and this could skew the data (Lund and Widmer, 1989).

Dao *et al* (1994) found that approximately one half (48.3%) of their myofascial pain dysfunction (MPD) patients experienced an increase in pain while one third (31.7%) reported a decrease following a similar chewing exercise. The authors suggested that opposing reactions to a chewing exercise (an increase versus a decrease in pain) could indicate the presence of two distinct pathologies. This study does not provide support for the hypothesis that two pathologies exist because of the much smaller percentage of subjects with a decrease in pain (3.7% - 15.4 % on the left and 3.7% - 3.8% on the right) in this study. The same material was chewed in both studies and the chewing time in this study was 4 minutes compared to the 3 minute exercise in the study by Dao *et al* (1994). It is unlikely that 1 additional minute of chewing could have resulted in the difference in results between the studies. This experiment shows that a brief chewing exercise is almost equally likely to increase a pain subject's pain (42.3% - 59.3%) as it is to evoke no change in pain (37.0% - 51.9%). It may have been beneficial to have patients chew for a longer period of time. Dao *et al* (1994) published preliminary data showing that prolonging the duration of the chewing limit to 15 minutes caused pain to increase progressively in time in some patients. It is possible that increasing the duration of chewing could have had an effect on the outcome of this study. However, the results of Tzakis *et al* (1994) suggested that this would not be the case since a 30 minute vigorous gum-chewing exercise caused pain their subjects but had no effect on their post-chewing EMG.

The results of this study provide little support for the hypothesis that there is a relationship between EMG activity and pain levels of pain subjects because of the low R^2 values, which ranged from 0 to 0.391. These low numbers indicated that the variability in EMG recordings was only partially due to the variability in pain scores. The findings from the regression analysis are not surprising because there were no consistent differences between the EMG activity of pain and non-pain groups even though 42.3% to 59.3% of the pain subjects reported an increase in pain after chewing compared to no increase in the non-pain subjects. This finding is supported by previous studies which have not found a reliable relationship between pain scores and EMG activity (Peck and Kraft, 1977; Dahlstrom *et al*, 1984; Burdette and Gale, 1988).

What, then, could be the theoretical explanation for pain during and after mastication? It is possible that pain during and following mastication is related to abnormal or unbalanced function of the muscles during mastication. Kumai (1993) used EMG to determine that TMD subjects chewed gum in an asymmetric pattern when compared to nonpain subjects. Stohler and Ash (1986) showed significantly prolonged contraction times and greater EMG amplitudes for chewing cycles with pain compared with pain-free chewing cycles. Dolan and Keefe (1988) found that high-pain TMD subjects had inappropriately higher activity in the left masseter when chewing gum on the right. This is contrary to the finding that the muscle activity is usually higher on the side that one is chewing. However, these EMG changes during mastication could only explain pain during the chewing exercise and would not explain pain after the chewing exercise, when the muscles had a chance to return to their baseline EMG levels.

Other explanations for pain during and after chewing have included morphologic and metabolic changes related to muscle contraction: a decrease in the muscle pH; an increase in the inorganic-phosphate-to-creatine-phosphate ratio; and an increase of intramuscular pressure (Dao *et al*, 1994). An additional hypotheses is that pain is related to the post-contraction blood flow (Jow and Clark, 1989). Lund *et al* (1991) suggested that hyperactivity or dysfunction is an adaptive response rather than the cause of pain. They offered a pain-adaptation model that explains the pain as a result of phasic modulation of excitatory and inhibitory interneurons supplied by high-threshold sensory afferents.

This study could have been improved by randomizing the subjects. Due to limitations in time, subjects were booked for the EMG recording appointment according to the order that they appeared for their screening appointment. All subjects were not screened on the same day due to time limitations and to the difficulties in recruiting subjects. In addition, the control subjects were unmatched for age to the experimental subjects. Thus, by using a randomized sample and a matched control group, the results of this study could have been strengthened.

This study included only female pain subjects within an age range of 18 to 40 years because more females present to clinicians asking for treatment (Rugh and Solberg, 1985). The limited age range was chosen because age-related changes in EMG recordings have been observed (Visser and De Rijke, 1974). If time had allowed the testing of a larger sample size, it would have been better to include subjects of different age groups and of a different gender so that the conclusions from this study could have been applied to the general population.

Considerable effort was made during this study to reduce the amount of stress for the subjects during all of the EMG recordings. Moller *et al* (1971) found that relaxation reduced the enhanced postural activity in pain patients. Both the "disregulation" and the "psychophysiological" theories state that the muscle hyperactivity is initiated centrally in the nervous system as a result of psychological stress (Katz *et al*, 1989). There is experimental evidence for the increased EMG activity in pain subjects compared with non-pain subjects during stressful stimuli (Yemm, 1969c; Mercuri *et al*, 1979; Dolan and Keefe, 1988; Katz *et al*, 1989). Perhaps the encouragement of relaxation and the listening to relaxing music during the baseline and chewing exercise may have reduced the postural EMG activity of the pain subjects. In addition, low EMG recordings in the clinical setting may not reflect typical responses to real life stressful circumstances (Cannistraci and Fritz, 1983).

This study could have been strengthened by narrowing the criteria for the selection of subjects in both the muscle pain and nonpain group. For example, bruxers were included in both the pain and nonpain groups. Sherman (1985) found that postural EMG activity was elevated in bruxers, whether or not they suffered from facial pain. However, Sherman is unclear in his description of how he determined that subjects were assessed for bruxism. He only indicated that they were included in a bruxism group if there was "physical evidence" of a history of clenching and bruxism. Studies showed that dental attrition, once thought to be physical evidence of bruxism, can be explained by a variety of other factors (Okeson, 1996). Today, bruxism habits are best assessed by using portable electromyography and sleep laboratories (Okeson, 1996). In the future, it might be useful to using one or more of these methods to screen for bruxism subjects when doing studies on postural EMG activity.

An improvement to this study could have been made by using a notch filter at 60-Hz, thus enabling the amplifiers to be set at a lower bandwidth. A bandwidth is defined as a functional characteristic generated by a filter circuit that eliminates unwanted frequencies and passes the desired frequencies (Peffer, 1983). A 30-Hz to 1000-Hz bandwidth is ideal where 60-Hz artifacts can be eliminated or are not paramount (Peffer, 1983). In this study, a bandwidth of 100-Hz to 1000-Hz was used because a notch filter could not be added to the amplifiers to filter out the 60-Hz signals. This is the same frequency signal as the power lines. It is possible that some low frequency signals were lost in the data collection as result of the bandwidth setting. In addition, it would have been ideal if the subjects' EMG activity was recorded over the entire duration of the study rather than samples during the study. Unfortunately, the computer was incapable of storing such massive amounts of data.

In fatigue studies, Hellsing and Lindstrom (1983) concluded that all members of a synergistic group of muscles should be simultaneously monitored because a reduced activity in one muscle may be compensated by an increased activity in another. The same may be true of studies that look at resting baseline activity. For example, medial pterygoid activity has been rarely studied because needle electrodes are required to measure its activity. The posterior temporalis is not often included in EMG studies for the same reason. It is possible that recording the synergistic activity of all jaw elevator muscles could give investigators a clearer picture of the postural activity before and after a chewing exercise.

A problem one encounters when comparing the results of the many studies in this area is a lack of consistency in criteria for choosing subjects, in the technology used, in the muscles recorded, etc. There is little standardization of the EMG apparati used in these studies. In addition, there is variability in how the EMG activity is measured. For example, some investigators measure normalized versus absolute EMG activity. There is variability in the definition of the study and control groups. Some studies have poor matching of their study and control groups in terms of age and gender. In addition, there is variability in the instruction given to the subjects during recording sessions. Finally, most studies do not control for bruxism due to the difficulty in assessing the presence of this parafunctional habit. All of these factors, and many more which are not listed, may contribute to the variability in results and the resulting diverse conclusions.

Although some significant results were found in the mean relative EMG activity of the left masseter between the groups, this magnitude of difference was small, the duration of this elevated muscle activity was low, and the elevation was not seen in all muscles. Thus, pain and non-pain female subjects showed a similar recovery to baseline EMG levels following a four minute chewing exercise. Linear regression analysis showed that very little of the variability in EMG activity was due to the variability in pain scores. Thus, from this research, it was concluded that there is no relationship between EMG activity and pain levels following a chewing exercise. This study does not support the "hyperactivity" theory as an etiology of TMD. In addition, this research does not support the hypothesis that biofeedback is effective by reducing muscle activity. However, this study does not preclude the use of biofeedback as a treatment modality.

3.2...Recommendations for Future Studies

1. The establishment of and adherence to a universal set of nomenclature would help avoid some of the confusion that occurs when results are being compared between studies on myogenous TMD patients. Progress has been made in this area by the International Headache Society (IHS).

2. It has often been stated that the presence of a bruxism habit in both myogenous TMD patients and nonpain controls can affect the baseline EMG activity (Sherman, 1985; Lund and Widmer, 1989; Lund *et al*, 1991). In future studies, it would be beneficial to identify these potential subjects using portable electromyography and/or sleep laboratories. These bruxers could then be excluded from EMG studies or else placed in a separate group of subjects in order to determine what effect bruxism has on resting EMG activity.

3. The monitoring of the entire synergistic group of mandibular elevator muscles in baseline EMG activity studies would help to establish an overall picture of how this group of muscles is behaving. Researchers would then be able to determine if certain muscles overcompensate when other muscles within the same synergistic group are hyper- or hypoactive. An example of a muscle that has not often been included in EMG studies is the medial pterygoid muscle. Due to present difficulties in placing needle electrodes, this may not be possible until further advancements in technology allow greater ease in muscle activity recording. For example, progress has been

made in the development of an intra-oral surface electrode, which measures the EMG activity of the medial pterygoid muscle.

4. Previous EMG research on TMD patients during mastication has indicated that there is evidence of unusual or unbalanced muscular function in the muscles of mastication compared with the chewing EMG data of control subjects (Kumai, 1993). Perhaps using biofeedback to show patients these imbalances during function could and help subjects to eliminate them. This would help determine if rectifying such imbalances improves TMD patients' pain condition elicited by a chewing episode. Such a study may help shed light on the exact mechanism of pain caused by chewing.

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Appendix

Item # 1: Information Sheet

A comparison of female muscle pain and asymptomatic subjects: electromyographic activity in masticatory muscles and pain response to masticatory exercise

Principal Investigator	
Dr. Paul Major	492-4469
Co-investigator	
Dr. Glenna M. Grykuliak	492-2398

Purpose: The purpose of this study is to compare the facial muscle activity of females (between the ages of 18 and 35) who have facial muscle pain to those who do not.

Procedures: You will be asked to visit us three times. All visits will be in the Dentistry/ Pharmacy building. First, you will be asked to fill out a questionnaire about muscle pain and to have a brief dental examination. The following two visits will be the data-gathering appointments for this study.

During the second session, the muscle activity of your facial muscles will be measured. This involves cleaning a few areas on your face with a facial cleansing solution. Nine sensors filled with conduction gel will be pasted to your face in several areas by adhesive tape. The sensors are connected to amplifiers that aid in measurement of the activity of the muscles. You will be able to see this activity on a screen. The recordings will take place before, during, and after a brief (4-5 minute) chewing exercise which you will perform with a soft piece of wax. In addition, you will rate any discomfort you feel both before and after the chewing exercise.

You will be asked to return for a second EMG recording session which will be identical to the first session described previously. It is preferable that this second session be completed during the same week as the first one. Each recording session will last about 30 minutes. Upon completion of the final session, you will be offered twenty dollars.

Possible Risks: All equipment is medically approved to be safe for human subjects. Similar studies have been conducted on other subjects and no negative side effects were reported.

Electromyography is considered to have virtually no side effects. It is possible that you may experience some excessive muscular activity such as cramps. In addition, you may have some unusual experiences such as a feeling of heaviness, a change in heartrate, a change in mood, or a headache following a recording session. Such side effects are extremely rare.

It is possible that you may experience an allergy to the conduction gel or adhesive tape although this is also extremely rare.

It is likely that some of the participants will experience discomfort during the chewing exercise. The chewing exercise, however, is brief. An identical chewing exercise has been performed in a recent Canadian study and no negative side-effects from this exercise were reported.

Confidentiality: Personal records relating to this study will be kept confidential. However, the data that is collected may be pooled with subsequent studies, enabling further research in this field.

Appendix

Item # 1: Information Sheet (Continued)

You are free to withdraw from the research study at any time. However, no money will be offered to anyone who does not complete the study. If any knowledge gained from this or any other study becomes available which could influence you decision to continue in this study, you will promptly be informed.

Please contact any of the individuals identified below if you have any questions or concerns:

Dr. Glenna Grykuliak, DDS, Orthodontic Graduate Student, University of Alberta 492-2398

Dr. Paul Major, DDS, Orthodontist, Professor and Director, TMJ Investigation Unit 492-4469

Appendix

Item # 2: Consent Form

A comparison of female muscle pain and asymptomatic subjects: electromyographic activity in masticatory muscles, and pain response to masticatory exercise

Principal Investigator: Dr. Paul Major (492-4469)
Co-investigator: Dr. Glenna Grykuliak (492-2398)

To be completed by the research subject:	Yes	No
Do you understand that you have been asked to be in a research study?	☐	☐
Have you read and recieved a copy of the attached information sheet?	☐	☐
Do you understand the risks involved in taking part in this research study?	☐	☐
Have you had an opportunity to ask questions and discuss this study?	☐	☐
Do understand that you are free to withdraw from this study at any time?	☐	☐
Has the issue of confidentiality been explained to you, and do you understand who will have access to your records?	☐	☐
Do you understand that the data collected for this study may be pooled with data from subsequent studies, enabling further research?	☐	☐

Who explained this study to you?____

____

I agree to take part in this study: YES ☐ NO ☐

Signature of Research Subject____

(Printed Name)____

Date:____________________

Signature of Witness____

Signature of Investigator or
Designee____

Appendix

Item # 3: Patient Questionnaire (Page 1)

Questionnaire SubjectName_______________

Please answer the following questions.

1. Please state your age:_________
2. Have you experienced any pain in your face within the last two to six months?
   ☐Yes ☐No
   (If you answered "no" to this question, please proceed to question # 5. Otherwise, continue on to the next question.)
3. How many times per week do you experience this pain?__________
4. For how many weeks or months have you experienced this pain?__________
5. Is one side of your face more painful than the other? ☐Yes ☐No
   If so, please indicate which one is more painful. ☐Right ☐Left

6. Have you ever experienced pain in your jaw joint on the sides of your face (including the areas in and around your ears)? ☐Yes ☐No
7. Have you ever noticed a "clicking" or "popping" sound in your jaw joints?
   ☐Yes ☐No
8. Has your jaw ever locked in an open or closed position? ☐Yes ☐No
9. Do you have a history of trauma to the head, including whiplash, blows to the face, or loss of consciousness? ☐Yes ☐No
   Briefly describe this trauma:____

10. Do you have chronic headaches or neck and shoulder pains? ☐Yes ☐No
11. Do you have frequent gastrointestinal disorders? ☐Yes ☐No
12. Do you grind or clench your teeth during the day? ☐Yes ☐No
13. Have you been made aware of clenching or grinding your teeth at night?
    ☐Yes ☐No
14. Do you wake up with an awareness of or about your teeth or jaws? ☐Yes ☐No
15. Do you have any awareness of the muscles of your neck and shoulders?
    ☐Yes ☐No
16. Do you have a tight or stiff neck? ☐Yes ☐No

Appendix

Item # 3: Patient Questionnaire (Page 1, Continued)

17. Are you taking any medications (i.e. muscle relaxants, anti-depressants) for your discomfort? ☐Yes ☐No
    If so, please list them:____
18. Are you currently wearing an acrylic splint or nightguard? ☐Yes ☐No
19. Have you ever had surgery on your jaw joint? ☐Yes ☐No

Appendix

Item # 4: Questionnaire (Page 2)

Subject Name__________________

Please put a checkmark beside the factors which cause or make your pain worse. You may choose more than one item:

Mastication ☐
Mouth Opening ☐
Yawning ☐
Mouth Closing ☐

Please indicate on a five-point scale to what extent your pain makes chewing difficult. You may circle the appropriate number.

0	Not at all
1	A little
2	Moderately
3	A lot
4	Extremely

Please state your preferred chewing side. Please circle the number that applies to you.

1	Both sides, without a preferred chewing side
2	Both sides, with a preferred RIGHT chewing side
3	Both sides, with a preferred LEFT chewing side
4	One side, always on the RIGHT
5	One side, always on the LEFT

Please rate your current pain intensity at rest on the Visual Analog Scale (on a scale from 0 to 100). You may mark anywhere on the line. The value of zero indicates no pain while the value of one-hundred indicates maximum pain. Please indicate separate scores for the right and left sides.

RIGHT ____
0 50 100

LEFT ____
0 50 100

Appendix

Item # 5: Pre-exercise Pain Score

Subject I.D._________
Session 1☐ 2☐

A comparison of female muscle pain and asymptomatic subjects: electromyographic activity in masticatory muscles and pain response to masticatory exercise

Pre-exercise Pain Score

Please rate your current pain intensity on the Visual Analog Scale (on a scale from 0 to 100). You may mark anywhere on the line. The value of zero indicates no pain while the value of one-hundred indicates maximum pain. Please indicate separate scores for the right and left sides. Please mark both lines even if pain on both sides is equal.

RIGHT ____
0 50 100

LEFT ____
0 50 100

Appendix

Item # 6: Post-exercise Pain Score

Subject I.D.________
Session 1☐ 2☐

A comparison of female muscle pain and asymptomatic subjects: electromyographic activity in masticatory muscles and pain response to masticatory exercise

Post-exercise Pain Score

Please rate your current pain intensity on the Visual Analog Scale (on a scale from 0 to 100). You may mark anywhere on the line. The value of zero indicates no pain while the value of one-hundred indicates maximum pain. Please indicate separate scores for the right and left sides. Please mark both lines even if pain on both sides is equal.

RIGHT ______________________________
0 50 100

LEFT ______________________________
0 50 100

Please state your preferred chewing side. Please circle the number that applies to you.

1 Both sides, without a preferred chewing side
2 Both sides, with a preferred RIGHT chewing side
3 Both sides, with a preferred LEFT chewing side
4 One side, always on the RIGHT
5 One side, always on the LEFT

Appendix

Item #7

Figures of Session 1 Mean Left MasseterEMG Activity vs Time (Pain and Non-pain)

Fig. 21: LM (Ses 1) Prior to Chewing

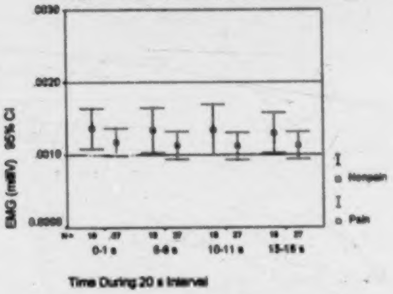


Fig. 22: LM (Ses 1) Immediately After

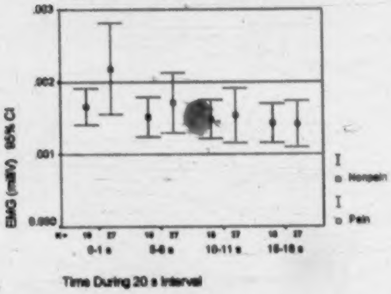


Fig. 23: LM (Ses 1) 1 Min After

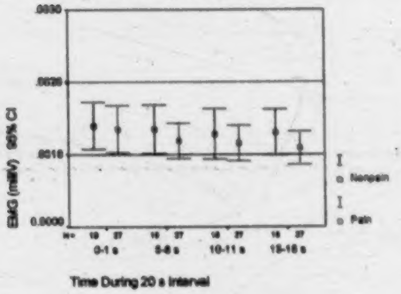
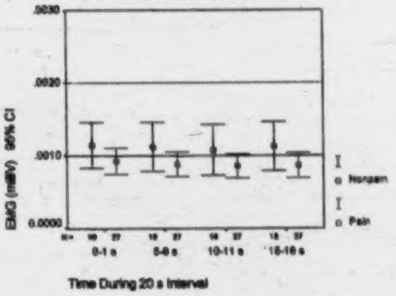


Fig. 24: LM (Ses 1) 3 Min After



Appendix
Item #8
Figures of Session 1 Mean Right Masseter EMG Activity vs Time (Pain and Non-pain)

Fig. 25: RM (Ses 1) Prior to Chewing

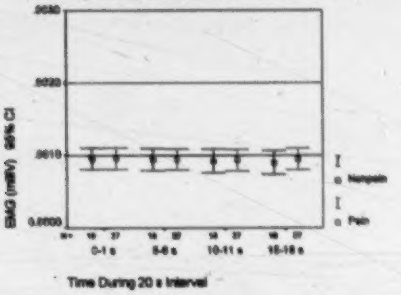


Fig. 26: RM (Ses 1) Immediately After

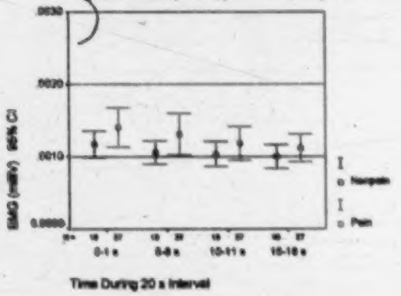


Fig. 27: RM (Ses 1) 1 Min After

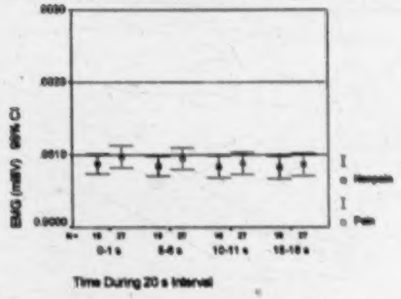
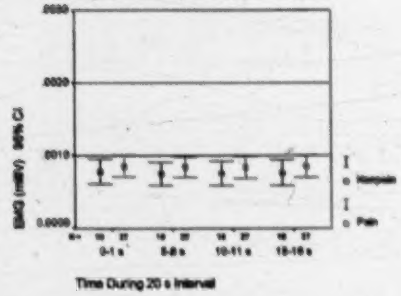


Fig. 28: RM (Ses 1) 3 Min After



Appendix
Item #9
Figures of Session 1 Mean Left Temporalis EMG Activity vs Time (Pain and Non-pain)

Fig. 29: LT (Ses 1) Prior to Chewing

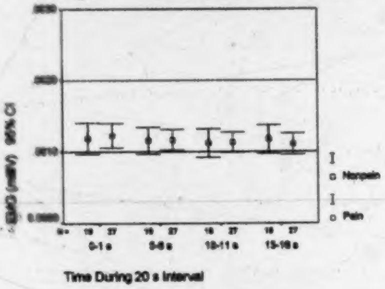


Fig. 30: LT (Ses 1) Immediately After

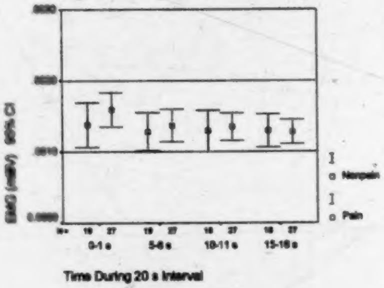


Fig. 31: LT (Ses 1) 1 Min After

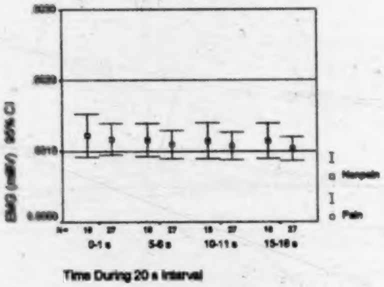
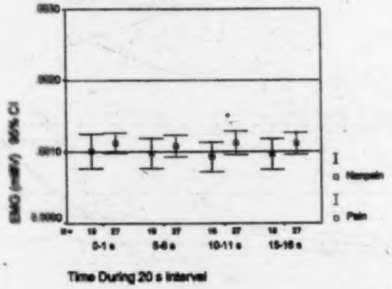
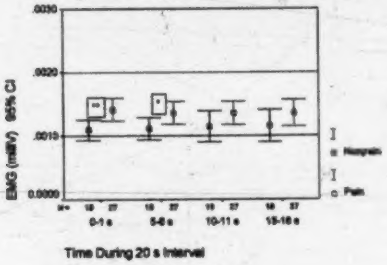


Fig. 32: LT (Ses 1) 3 Min After



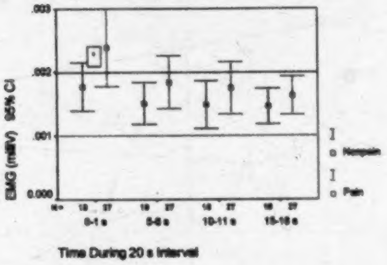
Appendix
Item #10
Figures of Session 1 Mean Right Temporalis EMG Activity vs Time (Pain and Non-pain)

Fig. 33: RT (Ses 1) Prior to Chewing



* = p-value < 0.05; ** = p-value < 0.01

Fig. 34: RT (Ses 1) Immediately After



* = p-value < 0.05

Fig. 35: RT (Ses 1) 1 Min After

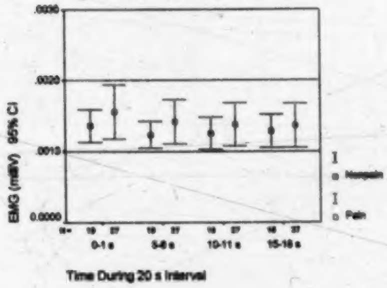
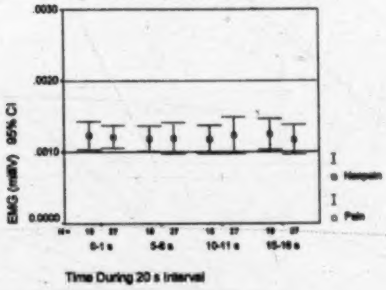


Fig. 36: RT (Ses 1) 3 Min After



Appendix Item #11. Table 3. P-Values of Left Masseter Mean EMG (Pain and Non-pain)

Time of Recording Relative to Chewing	Quartile 1 to 4	Session 1 vs. 2	Pain Rating Nopain vs. Pain	N	Mean (MicroV)	Standard Deviation (MicroV)	P-Value
Prior to	1	1	Nopain	19	1.36	0.564	
	1	1	Pain	27	1.18	0.486	0.121
	1	2	Nopain	20	1.06	0.432	
	1	2	Pain	25	1.22	0.385	0.124
	2	1	Nopain	19	1.34	0.64	
	2	1	Pain	27	1.13	0.486	0.102
	2	2	Nopain	20	1.05	0.373	
	2	2	Pain	25	1.22	0.388	0.078*
	3	1	Nopain	18	1.35	0.701	
	3	1	Pain	27	1.12	0.486	0.099*
	3	2	Nopain	20	1.03	0.343	
	3	2	Pain	25	1.18	0.425	0.095*
	4	1	Nopain	18	1.3	0.561	
	4	1	Pain	27	1.13	0.476	0.137
	4	2	Nopain	20	1.02	0.362	
	4	2	Pain	25	1.15	0.379	0.122
Immediately after	1	1	Nopain	19	1.67	0.523	
	1	1	Pain	27	2.19	1.59	0.062*
	1	2	Nopain	20	1.33	0.624	
	1	2	Pain	25	1.9	0.749	0.005***
	2	1	Nopain	19	1.52	0.576	
	2	1	Pain	27	1.72	1.06	0.212
	2	2	Nopain	20	1.25	0.603	
	2	2	Pain	25	1.8	1	0.02**
	3	1	Nopain	18	1.49	0.542	
	3	1	Pain	27	1.54	0.956	0.418
	3	2	Nopain	20	1.1	0.536	
	3	2	Pain	25	1.65	0.766	0.005***
	4	1	Nopain	18	1.43	0.542	
	4	1	Pain	27	1.42	0.813	0.461
	4	2	Nopain	20	1.07	0.525	
	4	2	Pain	25	1.53	0.67	0.006***
1 min after	1	1	Nopain	19	1.4	0.675	
	1	1	Pain	27	1.35	0.818	0.423
	1	2	Nopain	20	1.09	0.536	
	1	2	Pain	25	1.21	0.375	0.194
	2	1	Nopain	19	1.35	0.699	
	2	1	Pain	27	1.19	0.626	0.21
	2	2	Nopain	20	1.01	0.483	
	2	2	Pain	25	1.22	0.494	0.061*
	3	1	Nopain	18	1.29	0.7	
	3	1	Pain	27	1.16	0.619	0.269
	3	2	Nopain	20	0.925	0.44	
	3	2	Pain	25	1.16	0.386	0.031**
	4	1	Nopain	18	1.31	0.64	
	4	1	Pain	27	1.09	0.579	0.121
	4	2	Nopain	20	0.932	0.432	
	4	2	Pain	25	1.1	0.371	0.06*
3 min after	1	1	Nopain	19	1.14	0.656	
	1	1	Pain	27	0.928	0.457	0.112
	1	2	Nopain	20	0.941	0.44	
	1	2	Pain	25	1.14	0.549	0.099*
	2	1	Nopain	19	1.12	0.697	
	2	1	Pain	27	0.865	0.424	0.099*
	2	2	Nopain	20	0.923	0.403	
	2	2	Pain	25	1.08	0.47	0.127
	3	1	Nopain	18	1.08	0.702	
	3	1	Pain	27	0.86	0.42	0.122
	3	2	Nopain	20	0.968	0.445	
	3	2	Pain	25	1.03	0.343	0.298
	4	1	Nopain	18	1.13	0.675	
	4	1	Pain	27	0.87	0.435	0.079*
	4	2	Nopain	20	0.943	0.415	
	4	2	Pain	25	1.01	0.334	0.26

* = p-value < 0.1; ** = p-value < 0.05; *** = p-value < 0.01

Appendix Item #12. Table 4. P-Values of Right Masseter Mean EMG (Pain and Non-pain)

Time of Recording Relative to Chewing	Quartile 1 to 4	Session 1 vs. 2	Pain Rating Nopain vs. Pain	N	Mean (MicroV)	Standard Deviation (MicroV)	P-Value
Prior to	1	1	Nopain	19	0.956	0.31	
	1	1	Pain	27	0.959	0.383	0.489
	1	2	Nopain	20	0.84	0.411	
	1	2	Pain	25	1.02	0.369	0.063*
	2	1	Nopain	19	0.948	0.315	
	2	1	Pain	27	0.949	0.363	0.497
	2	2	Nopain	20	0.811	0.392	
	2	2	Pain	25	1.02	0.372	0.035**
	3	1	Nopain	18	0.926	0.331	
	3	1	Pain	27	0.938	0.376	0.457
	3	2	Nopain	20	0.788	0.377	
	3	2	Pain	25	0.966	0.379	0.044**
	4	1	Nopain	18	0.903	0.329	
	4	1	Pain	27	0.949	0.371	0.339
	4	2	Nopain	20	0.778	0.392	
	4	2	Pain	25	0.962	0.368	0.04**
Immediately after	1	1	Nopain	19	1.18	0.383	
	1	1	Pain	27	1.41	0.694	0.077*
	1	2	Nopain	20	1.08	0.577	
	1	2	Pain	25	1.35	0.59	0.062*
	2	1	Nopain	19	1.06	0.335	
	2	1	Pain	27	1.32	0.722	0.059*
	2	2	Nopain	20	9.86	0.485	
	2	2	Pain	25	1.17	0.43	0.088*
	3	1	Nopain	18	1.04	0.344	
	3	1	Pain	27	1.19	0.584	0.168
	3	2	Nopain	20	0.901	0.408	
	3	2	Pain	25	1.08	0.374	0.067*
	4	1	Nopain	18	1	0.335	
	4	1	Pain	27	1.12	0.479	0.19
	4	2	Nopain	20	0.889	0.409	
	4	2	Pain	25	1.07	0.353	0.055*
1 min after	1	1	Nopain	19	0.88	0.286	
	1	1	Pain	27	0.978	0.381	0.18
	1	2	Nopain	20	0.864	0.31	
	1	2	Pain	25	0.905	0.346	0.342
	2	1	Nopain	19	0.849	0.286	
	2	1	Pain	27	0.951	0.378	0.163
	2	2	Nopain	20	0.785	0.36	
	2	2	Pain	25	0.889	0.361	0.178
	3	1	Nopain	18	0.837	0.307	
	3	1	Pain	27	0.887	0.383	0.325
	3	2	Nopain	20	0.787	0.357	
	3	2	Pain	25	0.883	0.334	0.18
	4	1	Nopain	18	0.829	0.316	
	4	1	Pain	27	0.869	0.387	0.362
	4	2	Nopain	20	0.766	0.333	
	4	2	Pain	25	0.873	0.342	0.149
3 min after	1	1	Nopain	19	0.78	0.356	
	1	1	Pain	27	0.858	0.37	0.24
	1	2	Nopain	20	0.719	0.395	
	1	2	Pain	25	0.873	0.369	0.093*
	2	1	Nopain	19	0.748	0.338	
	2	1	Pain	27	0.844	0.359	0.184
	2	2	Nopain	20	0.719	0.403	
	2	2	Pain	25	0.841	0.323	0.133
	3	1	Nopain	18	0.753	0.339	
	3	1	Pain	27	0.841	0.379	0.217
	3	2	Nopain	20	0.726	0.438	
	3	2	Pain	25	0.826	0.32	0.2
	4	1	Nopain	18	0.785	0.363	
	4	1	Pain	27	0.853	0.382	0.222
	4	2	Nopain	20	0.711	0.406	
	4	2	Pain	25	0.819	0.322	0.163

* = p-value < 0.1; ** = p-value < 0.05; *** = p-value < 0.01

Appendix Item #13. Table 5. P-Values of Left Temporalis Mean EMG (Pain and Non-pain)

Time of Recording Relative to Chewing	Quartile 1 to 4	Session 1 vs. 2	Pain Rating Nopain vs. Pain	N	Mean (MicroV)	Standard Deviation (MicroV)	P-Value
Prior to	1	1	Nopain	19	1.19	0.448	
	1	1	Pain	27	1.23	0.437	0.388
	1	2	Nopain	20	0.932	0.319	
	1	2	Pain	25	1.19	0.455	0.02**
	2	1	Nopain	19	1.16	0.383	
	2	1	Pain	27	1.17	0.364	0.467
	2	2	Nopain	20	0.922	0.352	
	2	2	Pain	25	1.12	0.383	0.038**
	3	1	Nopain	18	1.13	0.398	
	3	1	Pain	27	1.14	0.348	0.465
	3	2	Nopain	20	0.92	0.386	
	3	2	Pain	25	1.11	0.388	0.052*
	4	1	Nopain	18	1.18	0.397	
	4	1	Pain	27	1.11	0.38	0.288
	4	2	Nopain	20	0.915	0.368	
	4	2	Pain	25	1.09	0.376	0.059*
Immediately after	1	1	Nopain	19	1.38	0.654	
	1	1	Pain	27	1.59	0.614	0.135
	1	2	Nopain	20	1.09	0.445	
	1	2	Pain	25	1.4	0.724	0.052*
	2	1	Nopain	19	1.28	0.557	
	2	1	Pain	27	1.37	0.584	0.308
	2	2	Nopain	20	1.13	0.705	
	2	2	Pain	25	1.42	1.21	0.174
	3	1	Nopain	18	1.3	0.585	
	3	1	Pain	27	1.35	0.489	0.363
	3	2	Nopain	20	0.994	0.391	
	3	2	Pain	25	1.32	1.08	0.103
	4	1	Nopain	18	1.3	0.471	
	4	1	Pain	27	1.28	0.43	0.453
	4	2	Nopain	20	0.965	0.364	
	4	2	Pain	25	1.27	0.96	0.097*
1 min after	1	1	Nopain	19	1.22	0.633	
	1	1	Pain	27	1.17	0.562	0.384
	1	2	Nopain	20	0.929	0.409	
	1	2	Pain	25	1.15	0.516	0.062*
	2	1	Nopain	19	1.16	0.487	
	2	1	Pain	27	1.1	0.494	0.332
	2	2	Nopain	20	0.886	0.363	
	2	2	Pain	25	1.16	0.544	0.028**
	3	1	Nopain	18	1.15	0.508	
	3	1	Pain	27	1.08	0.491	0.324
	3	2	Nopain	20	0.873	0.356	
	3	2	Pain	25	1.13	0.491	0.029**
	4	1	Nopain	18	1.15	0.497	
	4	1	Pain	27	1.03	0.423	0.212
	4	2	Nopain	20	0.899	0.362	
	4	2	Pain	25	1.11	0.472	0.057*
3 min after	1	1	Nopain	19	1.01	0.499	
	1	1	Pain	27	1.13	0.361	0.185
	1	2	Nopain	20	0.942	0.397	
	1	2	Pain	25	1.08	0.418	0.139
	2	1	Nopain	19	0.977	0.436	
	2	1	Pain	27	1.08	0.382	0.198
	2	2	Nopain	20	0.93	0.396	
	2	2	Pain	25	1.07	0.409	0.131
	3	1	Nopain	18	0.929	0.418	
	3	1	Pain	27	1.13	0.416	0.062*
	3	2	Nopain	20	0.932	0.39	
	3	2	Pain	25	1.03	0.382	0.204
	4	1	Nopain	18	0.968	0.434	
	4	1	Pain	27	1.12	0.387	0.118
	4	2	Nopain	20	0.924	0.392	
	4	2	Pain	25	1.03	0.377	0.185

* = p-value < 0.1; ** = p-value < 0.05; *** = p-value < 0.01

Appendix Item #14. Table 6. P-Values of Right Temporalis Mean EMG (Pain and Non-pain)

Time of Recording Relative to Clenching	Quartile 1 to 4	Session 1 vs. 2	Pain Rating Nopain vs. Pain	N	Mean (MicroV)	Standard Deviation (MicroV)	P-Value
Prior to	1	1	Nopain	19	1.09	0.336	
	1	1	Pain	27	1.42	0.453	0.006***
	1	2	Nopain	20	1.26	0.313	
	1	2	Pain	25	1.49	0.5	0.036**
	2	1	Nopain	19	1.12	0.363	
	2	1	Pain	27	1.36	0.448	0.025**
	2	2	Nopain	20	1.25	0.369	
	2	2	Pain	25	1.41	0.411	0.095*
	3	1	Nopain	18	1.15	0.492	
	3	1	Pain	27	1.36	0.457	0.077*
	3	2	Nopain	20	1.22	0.406	
	3	2	Pain	25	1.36	0.429	0.136
	4	1	Nopain	18	1.16	0.509	
	4	1	Pain	27	1.36	0.518	0.103
	4	2	Nopain	20	1.22	0.364	
	4	2	Pain	25	1.34	0.433	0.162
Immediately after	1	1	Nopain	19	1.77	0.788	
	1	1	Pain	27	2.39	1.56	0.042**
	1	2	Nopain	20	1.49	0.568	
	1	2	Pain	25	2.07	1.01	0.011**
	2	1	Nopain	19	1.51	0.68	
	2	1	Pain	27	1.84	1.05	0.118
	2	2	Nopain	20	1.51	0.871	
	2	2	Pain	25	1.82	1.11	0.15
	3	1	Nopain	18	1.49	0.755	
	3	1	Pain	27	1.75	1.04	0.186
	3	2	Nopain	20	1.31	0.632	
	3	2	Pain	25	1.75	1.2	0.075*
	4	1	Nopain	18	1.46	0.563	
	4	1	Pain	27	1.63	0.756	0.21
	4	2	Nopain	20	1.19	0.496	
	4	2	Pain	25	1.55	0.736	0.036**
1 min after	1	1	Nopain	19	1.36	0.473	
	1	1	Pain	27	1.56	0.961	0.182
	1	2	Nopain	20	1.2	0.52	
	1	2	Pain	25	1.44	0.644	0.094*
	2	1	Nopain	19	1.23	0.395	
	2	1	Pain	27	1.42	0.794	0.152
	2	2	Nopain	20	1.08	0.335	
	2	2	Pain	25	1.41	0.58	0.01***
	3	1	Nopain	18	1.25	0.454	
	3	1	Pain	27	1.38	0.771	0.25
	3	2	Nopain	20	1.08	0.375	
	3	2	Pain	25	1.34	0.52	0.029**
	4	1	Nopain	18	1.28	0.475	
	4	1	Pain	27	1.36	0.789	0.351
	4	2	Nopain	20	1.07	0.425	
	4	2	Pain	25	1.32	0.542	0.05**
3 min after	1	1	Nopain	19	1.23	0.414	
	1	1	Pain	27	1.22	0.412	0.443
	1	2	Nopain	20	1.09	0.363	
	1	2	Pain	25	1.31	0.623	0.072*
	2	1	Nopain	19	1.19	0.37	
	2	1	Pain	27	1.2	0.551	0.473
	2	2	Nopain	20	1.12	0.334	
	2	2	Pain	25	1.2	0.447	0.264
	3	1	Nopain	18	1.18	0.392	
	3	1	Pain	27	1.24	0.644	0.367
	3	2	Nopain	20	1.11	0.418	
	3	2	Pain	25	1.19	0.443	0.275
	4	1	Nopain	18	1.25	0.441	
	4	1	Pain	27	1.17	0.518	0.307
	4	2	Nopain	20	1.09	0.345	
	4	2	Pain	25	1.23	0.441	0.123

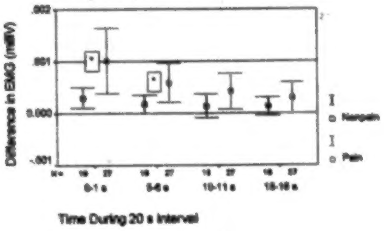
* = p-value < 0.1; ** = p-value < 0.05; *** = p-value < 0.01

AppendixItem # 15

Session 1 Differences Between Immediate Post-chewing EMG and Baseline EMG in the Masseter and Temporalis Muscles vs Time (Pain and Non-pain)

Figure 37

LM (Ses 1): Immed After - Baseline



* = p-value < 0.05

Figure 39

LT (Ses 1): Immed After - Baseline

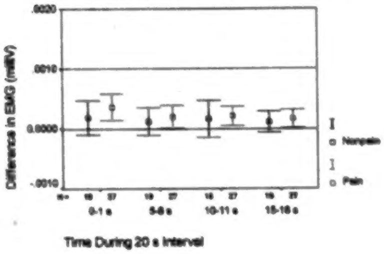


Figure 38

RM (Ses 1): Immed After - Baseline

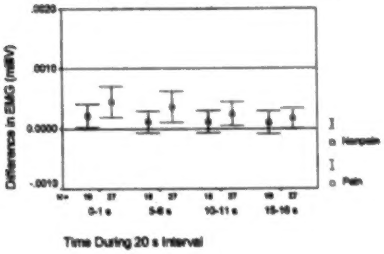


Figure 40

RT (Ses 1): Immed After - Baseline